AN ANALYTICAL STUDY TO EVALUATE THE RELATIONSHIP BETWEEN CENTRAL OBESITY AND BLOOD CHOLESTEROL LEVELS IN PATIENTS WITH PERIODONTAL DISEASE.

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CERTIFICATE

This is to certify that **Dr.B.Clement Roger**, Post graduate student (2008 – 2011) in the Department of Periodontology, Tamilnadu Government Dental College and Hospital, Chennai – 600 003 has done this dissertation titled AN **ANALYTICAL STUDY TO EVALUATE THE RELATIONSHIP BETWEEN CENTRAL OBESITY AND BLOOD CHOLESTEROL LEVELS IN PATIENTS WITH PERIODONTAL DISEASE** under our direct guidance and supervision in partial fulfillment of the regulations laid down by **The Tamil Nadu Dr.M.G.R. Medical University**, Chennai -600 032 for **M.D.S.,** (Branch – II) **Periodontology** degree examination.

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ABSTRACT

Background:

Obesity is a known risk factor for several chronic disease including hypertension, type 2 diabetes, dislipidemoia and coronary heart diseases.(must et al and modad et al 1999). Adipose tissue associated with obesity is known to secrete numerous proinflammatory cytokines such as IL-1 and TNF-α.these proinflammatory cytokines may play a crucial role in the close relationship among obesity, periodontitis and chronic diseases. (beck and offenbecher 2005)

Aim:

The aim of the present study was to determine whether obesity, which is associated with the development of number of chronic diseases, acts as a contributing factor in the development or progression of periodontal disease.

Materials and methods:

A total of 100 patients was include in the study with age group ranging from 18-59 years. They were divided into the younger 18-34 year olds and the middle aged 35-59 year old subjects. They were further subdivided based upon their BMI. The cutoff value for BMI was 23. Subjects WH ratio was also measured. Venous blood samples were collected for TC, TG, HDL, LDL and HsCRP. Periodontal parameters included were CAL, BI, PD were recorded in full mouth basis at 6 sites per tooth.

Results:

There was a positive correlation between increasing BMI andWH ratio values with the severity of CAL but the association was not statistically significant. TC, TG, HDL were vwithin the normal limits among the study subjects. There was a significant association between HsCRP in the younger age group whose BMI was 30.97±6.14.

Conclusion:

In this study, there was a positive correlation between indicators of obesity such as BMI and W:H ratio with the severity of periodontal disease but the association was not statistically significant. There may be a weak association between periodontitis and obesity. Further longitudinal studies are required to determine the association between obesity and periodontitis.

DECLARATION

TITLE OF DISSERTATION	**"An analytical study to evaluate the relationship between central obesity and blood cholesterol levels in patients with periodontal disease."**
PLACE OF STUDY	Tamil Nadu Government Dental College & Hospital, Chennai-600003
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NAME OF THE GUIDE	DR. MAHEASWARI RAJENDRAN
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I hereby declare that no part of the dissertation will be utilized for gaining financial assistance/any promotion without obtaining prior permission of the Principal, Tamil Nadu Government Dental College & Hospital, Chennai-600003. In addition, I declare that no part of this work will be published either in print or in electronic media without the guide who has been actively involved in dissertation. The author has the right to reserve for publish of work solely with the prior permission of the Principal, Tamil Nadu Government Dental College & Hospital, Chennai-600003.

Head of the Department **Guide** **Signature of the candidate**

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ABBREVATIONS

BMI - Body mass index

CAL - Clinical attachment level

CEJ - Cementoenamel junction

CHOL - Cholestrol

CRP - C-Reactive Protein

DHBS - 3,5-dichloro-2-hydroxybenzenesulfonic acid

GK - Glycerol kinase

H_20_2 - Hydrogen perioxide

HDL - High density lipoprotein

HDLD - High density lipoprotein detergent

HPO - Horseradish peroxidase

HsCRP - High senstive C-Reactive Protein

LDL - Low density lipoprotein

LPS - Lipo poly saccharide

PPD - Pocket probing depth

TG - Triglycerides

TNF- α - Tumour Necrosis Factor Alpha

VLDL - Very low density lipoprotein

W: H ratio - Waist Hip ratio

INTRODUCTION

Periodontitis is an inflammatory disease induced by bacterial biofilms that accumulate in the gingival margin, in which a series of aberrant inflammatory responses are initiated in the periodontal tissues **(Kornman *et al,* 1997)**. Interaction of the bacteria with the host results in an immuno-inflammatory response in the adjacent host tissues and triggers the release of numerous inflammatory mediators such as cytokines, some of which leads to the destruction of periodontal structures including supporting tissues of the teeth, alveolar bone and periodontal ligament.

Although bacterial pathogens are required to initiate the disease process, their presence alone is not sufficient to cause the tissue destruction that occurs in periodontitis **(William RC 1990, Birkedal – Hansen, 1993)**. The rate of progression may be modified by the presence of one or more risk factors such as smoking **(Bergstrom et al 2000),** diabetes mellitus **(Genco et al 1996)**, genetic disorders **(Hart TC et al 1994, Sofaer et al 1990)**, stress **(Genco and Grossi et al 1999)**, hyperlipidaemia **(Ozlan 2009)** and nutritional status **(Schifferle RE, 2005).**

Studies have shown that short term high fat diets results in prolonged impairment of polymorphonuclear leucocytes **(Cutler et al, 1999)**. Thus a hyperlipidaemic state may impair the host response to bacterial infections. Elevated serum lipids may directly interact with the macrophage cell membrane, interfering with membrane bound-receptors, enzyme systems and altering macrophage gene expression for essential polypeptides, growth factors and pro-inflammatory cytokines, such as TNF-α and IL-1β which are believed to be associated with periodontal disease **(Stashenko P et al, 1991, Heasman PA et al , 1993).**

Obesity on the other hand is a major public health problem . Obesity is a well known risk factor for several chronic diseases most notably hypertension, type 2 diabetes, dyslipidemia, and coronary heart diseases.**(Must et al 1999, Mokdad *et al* 2004, Fleagel *et al* 2006, Greg *et al* 2005)**. In fact, the adipose tissue secretes several cytokines and hormones collectively called adipokines **(Kershaw and Flier *et al* 2004)** involved in the inflammatory process, suggesting that similar pathways are involved in the pathophysiology of obesity and periodontitis.

Predominant cytokines secreted by adipocytes include TNF-α and IL-6 which are the main inducers of acute phase proteins such as the CRP **(Yudkin *et al* 2000)** which may be associated in the development of cardiovascular diseases **(American Heart Association,2000)**. Similarly periodontal pathogens affect local and systemic immune and inflammatory responses which may initiate systemic acute- phase response that in turn may **(Baumann H, Gauldi J, 1994)** lead to the production of inflammatory markers such as CRP and serum amyloid A protein.

Recent investigations suggested that even a moderate increase in CRP levels, such as those found in periodontitis patients, may predict a risk for atherosclerosis and CVD **(Tracy RP, LemaitreRN,et al, 1997, Haverkate F, Thompson S,et al 1997, Ridker PM, Cushman M, et al 1998, Lagrand, et al 1999) .**This study is aimed to evaluate whether obesity , blood cholesterol levels and high-sensitive C reactive protein has influence over the periodontal status and thereby to assess if obesity could be one among the potential risk factors for the pathogenesis of periodontal diseases.

AIM AND OBJECTIVES

The aim of the present study was to determine whether obesity, which is associated with the development of number of chronic diseases, acts as a contributing factor in the development or progression of periodontal disease.

For this purpose, the following objectives were undertaken

1. To evaluate the relationship between central obesity, blood cholesterol, hsCRP levels and periodontal disease.

2. To assess whether obesity is an independent risk indicator for periodontal diseases.

REVIEW OF LITERATURE

DEFINITION AND ASSESSMENT OF OBESITY:

The definition of obesity is based on the body mass index (BMI, also called Quetelet Index), which is the ratio of body weight (in kg) to body height (in m) squared **(Claude Lenfant et al,Expert Panel, 1998**, **National institute of health).**

BMI is highly correlated with fat mass and morbidity and mortality, and therefore sufficiently reflects obesity-related disease risk in a wide range of populations; however, there are some limitations. For example, for the same BMI, older persons tend to have a higher body fat composition. Furthermore, current BMI cut-off points for overweight and obesity are probably too high for Asian populations **(Choo et al, 2002**). More importantly, the BMI does not assess body fat distribution. It is well-known that abdominal (central, visceral, android) obesity, which is usually observed in men, is associated with a higher morbidity than the gluteofemoral (peripheral, gynoid) obesity typically observed in women **(Claude Lenfant et al, Expert Panel, 1998**, **National institute of health)**.

Body fat distribution is assessed by the measurement of waist circumference, with 102 cm in men and 88 cm in women, respectively, being the cut-off point for abdominal obesity associated with an increased risk of morbidity **(Claude Lenfant et al,Expert Panel, 1998**, **National institute of health)**. Waist circumference shows a close correlation with the amount of visceral adipose tissue, and visceral adipose tissue has been shown to be metabolically more active and secrete far greater amounts of cytokines

and hormones compared with subcutaneous adipose tissue **(Pouliot *et al.*, 1994; Wajchenberg, 2000; Berg and Scherer, 2005)**. Furthermore, a higher influx of portal fatty acids, cytokines, and hormones into the liver from omental adipose tissue may specifically distort hepatic metabolism, including abnormal lipoprotein synthesis, hepatic insulin resistance, and increased gluconeogenesis **(Eckel *et al.*, 2005; Haslam and James, 2005)**. Recently, large studies have indicated that measurement of waist circumference or waist-hip ratio may be a better disease risk predictor than BMI **(Wang *et al.*, 2005; Yusuf *et al.*, 2005).**

Classification and Definition of Overweight and Obesity established for non-Asian populations (based on Expert Panel, 1998, National institute of health)

Classification	**BMI (kg/m2)**
Underweight	< 18.5
Normal	18.5-24.9
Overweight	25.0-29.9
Obese - Class I	30.0-34.9
Obese - Class II	35.0-39.9
Obese - Class III	> 40

The recently proposed classification for Asian populations is:

(WHO/IASO/IOTF, 2000)

BMI < 18.5 => underweight;

18.5-22.9 => normal weight;

23.0-24.9 => overweight;

25.0-29.9 => obese class I;

> 30.0 => obese class II.

ROLE OF BODY FAT DISTRIBUTION:

A major predictor of health risk associated with obesity is body fat distribution. Body fat may be preferentially located in the abdomen (android obesity pattern) or surrounding the hips and thighs (gynoid obesity pattern). Numerous studies have demonstrated that the android pattern often reflects an accumulation of fat surrounding the abdominal visceral organs and is associated with a variety of metabolic derangements, including dyslipidemia, hypertension, and glucose intolerance. **(Kissebah AH *et al,* 1994)**. Thus, even at the same level of overweight, the individual with a greater amount of visceral fat is more likely to have, or to have developed, many of the serious health conditions associated with obesity. Sex **(Montague *et al,* 1997)** and ethnicity **(Stevens J.*et al*, 1995)** have an impact on body fat distribution and adipose tissue metabolism.

OBESITY-RELATED DISEASES:

Diseases for Which Sufficient Evidence Exists That Their Risk is Increased by Obesity (Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults—The evidence report. National Institutes of Health, 1998; Expert Panel, 1998; Haslam and James, 2005; Overweight, obesity, and health risk. National Task Force on the Prevention and Treatment of Obesity, 2000)

- Type 2 diabetes
- Hypertension
- Dyslipidemia
- Coronary heart disease
- Stroke
- Gall bladder disease
- Liver disease (non-alcoholic steatohepatitis)
- Musculoskeletal disease (osteoarthritis)
- Sleep apnoea and pulmonary dysfunction
- Cancer (colon cancer, endometrial cancer, post-menopausal breast cancer, kidney cancer)
- Reproductive abnormalities (menstrual irregularities, infertility)

Hypertension

Overweight and obesity have long been recognized as important determinants of elevated blood pressure levels **(Must *et al.*, 1999).** It is well-established that weight gain is consistently associated with increased blood pressure, and that weight loss decreases

blood pressure independent of changes in sodium intake. Compared with normal-weight individuals, Obese persons have an upto 5 times higher risk of hypertension, and upto 2/3 of cases of hypertension can be attributed to excess weight **(Wolf *et al.*, 1997)**. Mechanisms that have been implicated in the development of obesity-related hypertension include increased sympathetic nerve activity, sodium and volume retention, renal abnormalities, insulin resistance, hyperleptinemia, and increased secretion of angiotensinogen from adipocytes **(Kolanowski, 1999; Haslam and James, 2005)**.

Type 2 Diabetes:

The relationship between obesity and type 2 diabetes is particularly close. Obese persons have a more than 10-fold increased risk of developing type 2 diabetes compared with normal-weight persons **(Field *et al.*, 2001)**. Type 2 diabetes develops due to an interaction between insulin resistance and beta cell failure **(Stumvoll *et al.*, 2005)**. Several factors, including lipotoxicity and glucose toxicity as well as obesity derived cytokines, have been implicated in these processes **(Stumvoll *et al.*, 2005).**

Cardiovascular Disease and the Metabolic Syndrome:

Obese persons have an about 1.5-fold increased risk for cardiovascular disease (including coronary heart disease and cerebrovascular disease), and between 10 and 15% of all cases of cardiovascular disease can be attributed to overweight and obesity **(Wilson *et al.*, 2002)**.

The association with obesity is slightly stronger, and the population-attributable fraction (PAF, *i.e.*, the fraction of cases within the population that can be attributed to

overweight and obesity) is larger, for coronary heart disease (relative risk about 1.5 to 2.0; PAF 15 to 20%) than for cerebrovascular disease (RR 1.2 to 1.8; PAF 5 to 15%) **(Field *et al.*, 2001; Wilson *et al.*, 2002).**

Obesity is also associated with an about two-fold higher risk of heart failure and a 50% increased risk of atrial fibrillation **(Kenchaiah *et al.*, 2002)**.

The metabolic syndrome is a concept that encompasses metabolic abnormalities that co-occur to a greater degree than would be expected by chance alone, and which predispose individuals for a higher risk to develop cardiovascular disease **(Eckel *et al.*, 2005)**.

The metabolic syndrome is associated with components, namely, glucose intolerance, obesity, hypertension, and dyslipidemia (albeit the WHO also includes microalbuminuria as a component) (WHO, 1999; International Diabetes Federation, 2005). To date, most studies have used the definition provided in the **"Third Report of the National Cholesterol Education Program, Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) (NCEP-ATPIII)",** which requires the presence of at least three of the following metabolic abnormalities before the metabolic syndrome can be defined: **abdominal obesity, elevated triglycerides, reduced levels of HDL cholesterol, high blood pressure, and high fasting glucose.**

Although the exact underlying cause of the metabolic syndrome is unknown, the more recent definitions emphasize the focus on abdominal obesity as its core component **(International Diabetes Federation, 2005).** This approach is supported by a growing number of studies showing that the adipose tissue itself is capable of producing several

hormones and proteins, which are involved in the development of obesity related diseases.

Sleep Apnea and Pulmonary Dysfunction:

Sleep apnea is defined as a cessation of airflow during sleep for atleast 10 seconds, which can be central,obstructive , or mixed .Obesity is a major correlate with sleep apnea in men and women, among those having a BMI of at least 30 who are at greatest risk **(Strohl KP et al ,1998)**. Obesity is believed to change upper-airway geometry through loading of the wall of the pharynx or through increased deposition of periluminal fat . There are fairly well-established data indicating that symptoms of sleep apnea improve with weight loss. **(Strohl KP et al ,1998).**

Stroke:

Overweight may increase the risk for ischemic, but not hemorrhagic stroke. In the Nurses' Health Study, **(Rexrode KM *et al*, 1997)** ischemic stroke risk was 75% higher in women with a BMI of greater than 27 and 137% higher in those with a BMI of greater than 32

Gallbladder Disease:

The risk for gallstones and cholecystectomy increases with increasing body weight **(Khare M et al 1995)**

Liver Disease:

Nonalcoholic steatohepatitis (NASH) is a pathologic condition with all of the features of fatty infiltration, inflammation, and fibrosis seen in alcoholic liver disease, but occurring in the absence of excessive alcohol intake **(Sheth SG et al, 1997)**. It is more common in persons with obesity and with type 2 diabetes mellitus.

ASSOCIATION BETWEEN OBESITY AND PERIODONTAL DISEASE:

It has been suggested that obesity is second only to smoking as the strongest risk factor for inflammatory periodontal tissue destruction **(Nishida *et al.*, 2005)**. The first report on the relationship between obesity and periodontal disease appeared in 1977, when **Perlstein et al.** observed histopathologic changes in the periodontium in hereditary obese Zucker rats **(Zucker and Zucker, 1962; Perlstein and Bissada, 1977).** Using ligature-induced periodontitis, they found alveolar bone resorption to be greater in obese animals compared with non-obese rats **(Perlstein and Bissada, 1977).**

Also, it seemed that, under healthy oral conditions, obesity *per se* does not promote pathologic periodontal alterations; however, in response to bacterial plaque accumulation, periodontal inflammation and destruction were more severe in obese animals. In obese and hypertensive rats, plaque accumulation caused even more pronounced periodontal destruction than in obese animals, suggesting that a combination

of risk factors, such as those defined by the metabolic syndrome, elicit the most severe periodontal effects **(Koletsky, 1973; Perlstein and Bissada, 1977).**

Saito et al analyzed 241 healthy Japanese individuals and showed, for the first time, an association between obesity and periodontal disease in humans **(Saito et al., 1998)**. They applied the community periodontal index of treatment needs (CPITN) and estimated, based on their cross-sectional analysis, that the relative risk for periodontitis after adjustment for confounders such as age, gender, oral-hygiene status, and smoking was 3.4 in persons with BMI of 25 to 29.9 kg/m^2, and 8.6 in those with BMI above 30 kg/m^2.

In addition, studies have indicated that the fat distribution pattern plays a crucial role in the association with periodontitis **(Saito *et al.*, 2001; Al-Zahrani *et al.*, 2003; Wood *et al.*, 2003).** Saito *et al.* found, in 643 healthy Japanese adults, that high upper body obesity and high total body fat were correlated with a higher risk of periodontal disease, compared with normal-weight persons **(Saito *et al.*, 2001)**.

An examination of the NHANES III data demonstrated that waist-to-hip ratio, BMI, fat-free mass, and log sum of subcutaneous fat significantly correlated with periodontal disease **(Wood *et al.*, 2003).**

Also, high waist circumference was especially associated with periodontal disease in 18- to 34-year-old persons, but not in older adults, suggesting a closer correlation between high waist circumference and periodontitis in young adults **(Al-Zahrani et al., 2003)**.

In 706 South Brazilian individuals, no correlation between BMI and periodontal disease was found in men, but a strong correlation was found in obese females **(Dalla Vecchia et al., 2005).**

Another recent study by Saito *et al.* concluded that obesity is associated with deep periodontal pockets, independent of glucose tolerance status **(Saito et al., 2005).**

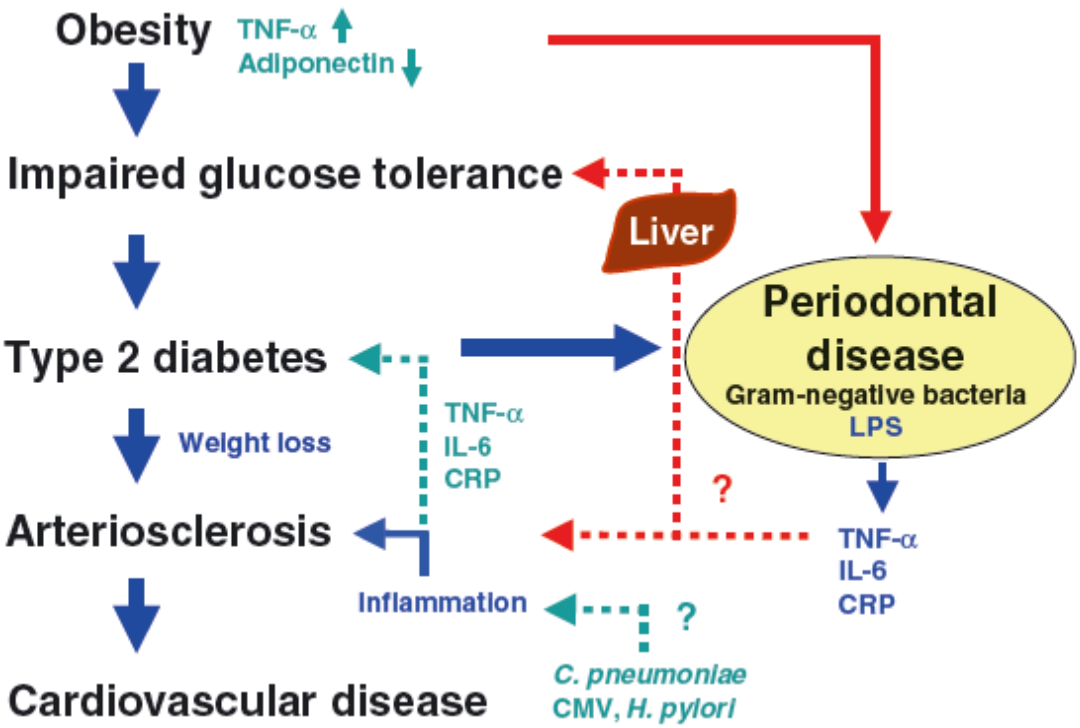
Genco et al. analyzed NHANES III data and demonstrated that BMI was positively correlated with the severity of periodontal attachment loss; they found that this relationship is modulated by insulin resistance **(Genco et al., 2005).**

Recent studies have indicated that maintaining a normal weight by regular physical activity is associated with a lower periodontitis prevalence **(Wakai et al., 1999; Karjalainen et al., 2002; Merchant et al., 2003; Al-Zahrani et al., 2005a,b).**

Individuals who pursued regular exercise have lower plasma levels of inflammatory markers, such as IL-6 and C-reactive protein (CRP), and show an increased insulin sensitivity that may beneficially affect periodontal health **(Merchant et al., 2003; Pischon et al., 2003; Al-Zahrani et al., 2005a).**

A study that analyzed the NHANES III study population demonstrated that individuals who maintained a normal weight, pursued regular exercise, and consumed a diet in conformity with the Dietary Guidelines for Americans and the Food Guide Pyramid recommendations were 40% less likely to have periodontitis **(Al-Zahrani et al., 2005).**

ASSOCIATION OF PERIODONTITIS WITH OBESITY-RELATED CHRONIC DISEASES:



Pro-inflammatory cytokines may play a crucial role in the close relationship among periodontitis, obesity, and chronic diseases **(Beck and Offenbacher, 2005; Genco et al., 2005)**. In fact, this association may be multidirectional. For example, it has been well-established that inflammation is an essential component in the development of atherosclerosis, and observational studies showed that periodontitis is associated with a moderately, but significantly, higher risk of coronary heart disease **(Beck and Offenbacher, 2005; Dietrich and Garcia, 2005; Mattila et al., 2005).**

Inflammatory diseases like periodontitis induce the production of pro-inflammatory cytokines such as TNF-α, IL-1, and IL-6 **(Beck et al., 1996; Loos, 2005)**. It has been suggested that the secretion of TNF-α by adipose tissue triggered by LPS

from periodontal Gram negative bacteria promotes hepatic dyslipidemia and decreases insulin sensitivity **(Saito et al., 2001; Nishimura et al., 2003)**.

Type 2 diabetes and decreased insulin sensitivity are associated with the production of advanced glycation end-products (AGE), which trigger inflammatory cytokine production, thus predisposing for inflammatory diseases such as periodontitis **(Grossi and Genco, 1998; Genco et al., 2005)**. These observations suggest a potential interaction among obesity, periodontitis, and chronic disease incidence, although present studies are insufficient to conclude whether such associations are causal. Thus, in addition to being a risk factor for type 2 diabetes and coronary heart disease, obesity-related inflammation may also promote periodontitis. Conversely, periodontitis, once it exists, may promote systemic inflammation and thereby increase the risk of coronary heart disease **(Beck and Offenbacher, 2005; Loos, 2005).** In this context, it is interesting to note that periodontal treatment has been shown to reduce circulating TNF-α and serum levels of glycosylated hemoglobin, and has beneficial effects on the control of type 2 diabetes **(Grossi and Genco, 1998)**.

Adipose-tissue-derived hormones and cytokines (adipokines):

The traditional view of adipose tissue as a passive reservoir for energy storage is no longer valid. Adipose tissue is now known to express and secrete a variety of bioactive peptides, known as adipokines, which act at both the local (autocrine/paracrine) and systemic (endocrine) level. It is now clear that adipose tissue is a complex and highly active metabolic and endocrine organ. In addition to adipocytes, adipose tissue contains fibroblasts, preadipocytes, tissue resident macrophages, and vascular constituents. Macrophages are known to be crucial contributors to inflammation, but more recently, it

has been recognized that adipocytes demonstrate significant intrinsic inflammatory properties as well. Like macrophages, the adipocyte is exquisitely sensitive to infectious disease agents and cytokine-mediated inflammatory signals; it expresses a host of receptors, enabling it to sense the presence of pathogens and inflammation, and on stimulation of these receptors, it activates multiple inflammatory signal transduction cascades, that induces and secretes a number of potent inflammatory cytokines and acute phase reactant proteins.

The underlying biological mechanisms for the association of obesity with periodontitis are not well-known; however, adipose-tissue-derived cytokines and hormones may play a key role. Fat tissue is not merely a passive triglyceride reservoir of the body, but also produces a vast amount of cytokines and hormones, collectively called adipokines or adipocytokines **(Kershaw and Flier, et al 2004)**, which in turn may modulate periodontitis.

Adipose tissue secretes pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6). TNF-α and IL-6 are the main inducers of acute-phase hepatic protein production, including C-reactive protein (CRP) **(Yudkin et al., 2000).**

Both TNF-α and IL-6 have been shown to impair intracellular insulin signaling, which may lead to insulin resistance **(Hotamisligil, 2000; Rotter *et al.*, 2003)**. In humans, plasma levels of TNF-α, IL-6, and CRP are closely related to obesity and insulin resistance **(Hotamisligil, 1999; Kern et al., 2001)**.

There is compelling evidence that inflammation plays an essential role in the development of type 2 diabetes mellitus and atherosclerosis, and studies in humans

suggest that circulating inflammatory marker levels may predict type 2 diabetes and cardiovascular events years in advance of the onset of these diseases **(Pradhan et al., 2001; Libby, 2002; Pradhan and Ridker, 2002; Ridker, 2002; Danesh et al., 2004; Pai et al., 2004).**

An up to 10-fold increase in local and systemic expression of inflammatory cytokines, such as TNF-α and IL-6, by monocytes and macrophages has been reported in some individuals with periodontitis **(Beck et al., 1996).**

In persons with periodontitis, bacterial pathogens, endotoxins, and inflammatory cytokines may systemically trigger an up-regulated leukocytosis, synthesis of acute-phase proteins (CRP, Amyloid A), and enhanced lipid metabolism, along with increased serum cholesterol and triglyceride levels, which may contribute to the risk of systemic diseases such as cardiovascular diseases **(Beck et al., 1998; Loos et al., 1998; Nishimura et al., 2003; Beck and Offenbacher, 2005; Loos, 2005; Mattila et al., 2005).**

Acute –phase proteins:

It has been long established that immune mediators originating from a site of infection or from a site of severe trauma may activate hepatocytes in the liver to produce large quantities of Acute –phase proteins **(Steel et al, 1994).** The acute-phase response is characterized by fever, increased vascular permeability, and a general elevation of metabolic processes**.** These acute-phase reactants posses a wide variety of functions. These include pro-inflammatory properties, activation of complement factors, neutralization of invasive pathogens, stimulation of repair and regeneration of variety of tissues **(Steel et al, 1994).** The acute –phase proteins receiving most attention includes C-

reactive protein (CRP), serum amyloid P component, serum amyloid A protein and α 1 – acid glycoproteins (AGP).

C-Reactive Protein:

C-reactive protein (CRP) is an acute-phase reactant produced by the liver in response to diverse inflammatory stimuli, including heat, trauma, infection, and hypoxia **(Pepys and Baltz, 1983)**. In patients with overwhelming systemic infection, serum levels of CRP can exceed 100 mg/L, providing a useful marker for tracking the course of the infection.

Historically, CRP values of > 10mg/l have been regarded as diagnostic for a bacterial infection, while values <10mg/l have been neglected (also because one was unable to measure CRP accurately <10mg/l). in the mid 1990s, hi-sensitivity assays for CRP came into widespread use, allowing laboratories to determine CRP levels as low as 0.3 mg/l.

However, the clinical relevance of much smaller increases in CRP has been highlighted recently in epidemiological studies demonstrating that individuals with "high-normal" values of CRP have increased risks for chronic diseases that have an inflammatory basis, including cardiovascular disease. Established risk factors for "high-normal" values of CRP within the general population include older age, cigarette smoking, chronic bacterial infections, and chronic bronchial inflammation **(Palosuo et al., 1986; Saikku et al., 1992; Patel et al., 1995; Mendall et al., 1996; Ridker et al., 1997)**.

CRP has been shown to play a role in the pathogenesis of atherosclerosis through different mechanisms including binding the phosphocholine of oxidized low density lipoproteins, upregulating the expression of adhesion molecules in endothelial cells, increasing low density lipoprotein uptake in to macrophages, inhibiting endothelial nitric oxide synthase expression in aortic endothelial cells and increasing plasminogen activator inhibitor – 1 expression and activity. (**Black, Kushner et al 2004).**

The first observations of the well-known association between CRP and cardiac risk was in 1954,when it was found that after myocardial infarction, there was a dramatic rise in circulating CRP levels and the amplitude of this rise correlated with poor prognosis. **(Kroop IG, Shackman NH. 1954, Anzai T, Yoshikawa T, Shiraki H).**

Subsequently, it was also found that pre-infarct elevated CRP levels correlated with an increased risk of future cardiac events as well. Not only were pre-infarction CRP levels correlated with increased risk for later adverse cardiac events and sudden death, elevated circulating levels of IL-6, IL-18, levels have been correlated recently with increased risk of congestive heart failure development in elderly patients even before evidence of CVD. **(Vasan RS, Sullivan, 2003).**

Numerous additional studies have further strengthened the association of elevated CRP levels with nearly all the important cardiovascular risk factors, including insulin resistance and diabetes, metabolic syndrome, hypertension, smoking, and dyslipidemia **(Saito M, Ishimitsu T, 2003)**. Elevated CRP levels in obese patients are not only prognostic for the development of CVD but also predictive of the risk of progression to type 2 diabetes mellitus. (**Pradhan AD, Manson JE, 2001**).

Elevated CRP levels in obesity and the decrease in its levels associated with weight loss provide another suggestive link between CRP and obesity-associated risks for CVD and diabetes. (**Heilbronn LK, Noakes, 2001; Kopp HP, Kopp CW, Festa A, 2003; Tchernof A, Nolan A, 2003; Esposito K, Pontillo A, 2003).**

PERIODONTAL DISEASE AND CRP:

An ever-increasing number of reports have demonstrated CRP in relation to periodontitis.

Ebersole et al, 1996 observed relatively high levels of CRP: 9 mg/1 in study group versus 2 mg/1 in controls.

Hujoel et al, 2001 reported a median CRP of 2 mg/1 was observed for periodontitis patients, while the median among controls was 0 mg/l**.**

Loos et al, concluded that the highest CRP values were found in patients with a generalized form of periodontitis (median 1.45 mg/1); for patients with a more localized form of periodontal disease, the median CRP value was 1.30 mg/1, while healthy controls presented with a median of 0.90 mg/1 .This demonstrates that CRP also behaves in a dose-dependent manner.

Epidemiological studies from **Buffalo, New York,** reported elevated CRP in blood plasma among subjects with severe periodontal attachment loss in comparison to individuals with minimal to no attachment loss (**Noack, Genco et al, 2001, Glurich, Grossi et al, 2002).** These studies also reported that those subjects who were infected with periodontal pathogens had clearly higher levels of CRP than those who were not harboring them.

Buhlin et al 2003 observed higher plasma levels of CRP in severe periodontitis patients in comparison to healthy controls.

Recently elevated CRP concentrations were found among periodontal patients in New York City of Hispanic, Asian, or African American background. In this study by **Craig et al, 2003,** a dose response effect of CRP was found: the numbers of periodontal lesions were positively correlated to CRP values.

Cross-sectional epidemiological surveys in Japan revealed that individuals with the highest CPITN or community periodontal index (CPI) scores had higher CRP values than those with lower CPITN or CPI scores. (**Wakai et al 1999, Takami et al, 2003).**

In another Japanese study among men only, a strong positive correlationwas found between alveolar bone loss and plasma CRP level. (**Saito et al, 2003).**

In a cross-sectional epidemiological survey in the United states, **Slade et al, 2000** observed CRP values of 4.5 mg/l for subjects with >10% pockets of >4 mm in depth in contrast to those subjects without pockets >4 mm, who had an of average 3.3 mg/l CRP.

Some intervention studies have investigated the effects of periodontal therapy on CRP levels. **Mattila et al (2004)** reported a reduction of CRP concentrations on 30 patients with chronic periodontitis; the median value at baseline was reduced from 1.05 mg/1 to 0.7 mg/1 after therapy. This reduction of CRP levels was most clearly observed among those who had CRP values >2 mg/1 before therapy.

Recently, a large pilot intervention study among periodontitis patients was reported in which 94 patients with severe periodontitis received non-surgical therapy. At 6 months after treatment, the median value for CRP was reduced by 0.5 mg/1 (1.9 mg/1 at baseline and 1.4 mg/1 6 months after basic periodontal therapy). Interestingly, two

months after therapy, the reduction in CRP was not yet observed, **(Daiuto et al, 2004)** suggesting a slow healing process in these subjects with severe periodontitis. Similar observations were made by **Ide et al (2004)** while **Mattila et al.(2004)** observed their results only 6 weeks postoperatively. Further intervention studies are needed to completely understand the dynamics of CRP in relation to periodontal therapy.

PERIODONTITIS AND HYPERLIPIDEMIA:

Animal studies have demonstrated a causal relationship between hyperlipidemia and periodontitis. Investigations in rats by **Ueno et al in 1965** have demonstrated that animals placed on high fat diet developed periodontitis.

The study of **Feingold et al 1992** showed that the administration of low doses of endotoxins in rats resulted in hypertriglyceridemia suggesting the presence of a similar response in local infections such as periodontal disease.

The study of **Memon et al 1993** has proved that induction of periodontitis by P.gingivalis in rats resulted in increased level of TGL. Using similar methodology, the same result was observed in work of **Doxey et al in 1998.**

Studies in non human primates by **Ebersole et al in 1999** have demonstrated elevations in serum lipids of inflammatory biomarkers and LDL/TG during gingivitis and periodontitis. Additionally these changes were exacerbated by high fat diet and animals on high fat diet exhibited more severe periodontitis.

Human studies appear to confirm the hyperlipidemia - periodontitis relationship. Subjects with hyperlipidemia demonstrated significantly more severe periodontitis than community based controls and the degree of periodontal breakdown was positively correlated to plasma lipid levels. (**Pohl et al 1995)**. A study measuring serum lipid levels in systemically healthy patients with moderate periodontitis by **Netea et al in 1997** showed a significant elevation of serum LDL/TG in periodontitis patients compared to controls.

Studies involving clinical evaluation/laboratory assessment of healthy and diabetic patients suggest a potential linkage between periodontitis, elevated serum lipid and level of bacteremia. (**Cutler et al 1999)** . Analysis of sera from random healthy clinic patients demonstrated significant elevations of serum lipids and P.gingivalis titers in patients with periodontitis compared to patients without periodontitis. It also demonstrated significant association between the presence of disease with total serum cholesterol, TG and amount of P.gingivalis antibodies. (**Cutler et al 1999)**.

According to National Cholesterol Education Program periodontal disease was more prevalent in subjects with a serum HDL-C concentration <60mg/dl suggesting that periodontal disease is exacerbated by metabolic syndrome.(**NCEP 2000)**.

Losche et al in 2000 showed that the plasmatic level of lipids in periodontitis patients were significantly higher than the healthy individuals.

Noack et al in 2000 studied 100 subjects and suggested that hyperlipidemia is a risk factor for periodontal disease, while impaired glucose tolerance is not.

In **2002, Katz et al** evaluated the periodontal health of over 10000 Israeli military service men and women and compared the results with their blood lipid levels and did not find a significant association between the presence of periodontal pockets and high levels of triglycerides. While some authors like **Katz et al 2001, 2002** mentioned that there were significant correlations between periodontal status and cholesterol levels, whereas **Cutler et al 1999, Morita et al 2004 ,** indicate that there were significant association between trigyceride level and periodontal disease.

After periodontal treatment, significant increase in serum HDL-C concentration, HDL2/HDL3 and HDL phospholipids content were observed. In this study, **Pussinen et al 2004** stated that periodontitis decreases the antiatherogenic potency of HDL.

A study by **Aki Izumi et al on 2009** showed that higher total cholesterol is associated with lower prevalence of periodontitis in non smoking elderly patients.

MATERIALS AND METHODS

STUDY DESIGN AND SUBJECT SELECTION:

The study was approved by the Institutional Ethical committee. 100 patients in the age group of 18- 59 years who attended the Out patient department , Department of Periodontics, Tamil Nadu Government Dental College, Chennai between June and October 2010 participated in the study .

The study population was categorized into two groups based on their age; the younger age group comprising 50 subjects between the ages 18-34, and the middle aged group comprising 50 subjects between the ages 35-59. Patients more than 59 years were not included in the study due to the expected presence of many confounding factors, extreme age being one of it and the presence of many systemic illnesses. These two groups were designated as Group I and Group II respectively.

Subjects within these two groups were further sub-divided into 2 subgroups depending on their body mass indices. One subgroup comprised of 25 subjects each. The BMI cut off decided to categorize the subjects was 22.9 which is accordance to the guidelines endorsed by the INDIAN DIABETIC ASSOCIATION for the Indian population who are at increased risk for developing overweight/obesity related complications compared to their western counterparts. Hence one subgroup consisted of subjects having BMI 22.9 or less and the other subgroup comprised of subjects with BMI 23 or more.

Similarly the 2nd group comprising of age range 35-59 years was also divided in the same way into 2 subgroups. Both males and females participated in the study irrespective of gender.

To summarize, the subjects were divided in the following way

GROUP I: Age range from 18-34 years

GroupIa: subjects having BMI 22.9 or less

GroupIb: subjects having BMI 23 or more

GROUPII: Age range from 34-59 years

GroupIIa: subjects having BMI 22.9 or less

GroupIIb: subjects having BMI 23 or more

A written informed consent was obtained from all patients. A complete history and clinical features of the subjects attending study were obtained. Prior to blood sample collection , plaque index, bleeding index, probing depth, clinical attachment loss were recorded.

5 ml of fasting venous blood samples were collected from the patients for the estimation of lipid profile and CRP levels.

INCLUSION CRITERIA:

1. Patients willing for voluntary participation and having signed the informed consent.
2. Age : 18-59 years
3. Gender : Both males and females
4. Patients with minimum 20 teeth.

EXCLUSION CRITERIA:

1. Trauma or tooth extraction within 2 weeks
2. Patients drug history (anticholesterol drugs, antibiotics, anticoagulants) for past 2 months.
3. Any chronic infectious diseases.
4. Pregnancy
5. Anemia, bleeding disorders or any other hematological disorder
6. Cardiac or respiratory diseases
7. Stroke or diabetes
8. Smoking habits, pan chewing, alcoholism
9. Malnourished or underweight patients.
10. History of periodontal treatment 6 months prior to study.

STUDY PROTOCOL:

1 Institutional ethical committee approval
2. Medical History and Informed Consent
3. Periodontal Examination using clinical parameters namely, Bleeding Index, Plaque Index, Probing Pocket Depth and Clinical Attachment Level.
4. Measurement of body mass index and waist hip ratio
5. Collection of blood samples
6. Estimation of serum blood lipid profile and HsCRP.

Following selection of subjects, written informed consent was obtained from all the subjects selected for the study after explaining the study procedure. Examination was preceded by a thorough medical and dental history of the subjects. Intra-oral examination was done using mouth mirror and William's Periodontal Probe. Periodontal Evaluation was done by measuring the Bleeding Index, Plaque Index, Probing Pocket Depth (PPD) and Clinical Attachment Level (CAL).

ARMAMENTARIUM:

For examination:

Mouth mirror

William's periodontal probe

Gloves and mask

Sample collection:

5ml syringe

Spirit

Sterile cotton

To measure general parameters:

Weighing machine

Height indicator

Measuring tape

Reagents/ Consumables:

SYNCHRON CX MULTI Calibrator

CHOL Reagent Cartridges

SYNCHRON CX9 **PRO** TG Reagent Cartridges

Sekisui HDL Reagent

SYNCHRON CX reagent cartridges

Sekisui Cholestest Calibrator

Sekisui LDL Reagent

Cuvettes & Tips

Equipment:

Synchron CX9 PRO Clinical Chemistry Analyzer

General Examination:

Waist-hip Ratio:

Subjects' waists and hips were measured. Upper body obesity is defined as a waist-hip ratio of ≥0.8 for females and ≥0.9 for males. These differences were derived from skeletal differences and differences in fat distribution between sexes. The difference was 0.12 between the sexes and was almost the same in any age categories.

Measurement of W: H ratio:

A measuring tape was used to measure the waist and hips. Waist measurement was done at the region of the umbilicus and the hips are measured at its widest position.

Normal W: H Ratio

Females <0.8

Males <0.9

Height measurement: Height was measured using a height indicator in cms.

Weight measurement: measured using weighing machine in kgs.

$$\text{BMI:} \quad \frac{\text{weight in kg}}{\text{Height in meter}^2}$$

CLINICAL PARAMETERS:

GINGIVAL BLEEDING INDEX (Ainamo & Bay 1975):

Teeth examined - All teeth

Surfaces examined - 4 sites for each tooth (Mesial, buccal, Distal, lingual,)

The presence or absence of bleeding is determined by gentle probing of the gingival crevice with a periodontal probe.

Criteria for Scoring:

Positive score (+) - Presence of bleeding within 10 seconds

Negative score (-) - Absence of bleeding

$$\text{\% of bleeding sites} = \frac{\text{Total number of positive score}}{\text{Total number of surfaces of all teeth}} \times 100$$

PLAQUE INDEX (Silness and Loe 1964):

Teeth examined – All teeth

Surfaces examined – 4 sites for each tooth (disto-facial, Facial, Mesio-facial, lingual/palatal)

Criteria for Scoring:

Score 0 - No plaque

Score 1 - Plaque not visible to the naked eye, detected by explorer

Score 2 - Thin to moderate accumulation of soft deposits within the gingival pocket or on tooth, visible to the naked eye

Score 3 - Abundance of soft matter within gingival pocket or on tooth surface and margin, inter-dental area stuffed with soft debris

Calculation :

Plaque index for a tooth= total score from 4 areas/4

Plaque index for the individual = Total Plaque indices for all teeth / No. of teeth examined.

Interpretation: Score 0 – Excellent oral hygiene

0.1 to 0.9 – Good oral hygiene

1.0 to 1.9 – Fair oral hygiene

2.0 to 3.0 - Poor oral hygiene

PROBING POCKET DEPTH (PPD) (Grant 1965):

Probing Pocket Depth was measured from the gingival margin to the base of the pocket using William's Periodontal Probe. The probe was passed within the gingival sulcus along the circumference of the tooth.

Three measurements were made on the buccal aspect and three on the lingual aspect of each tooth – total of six sites per tooth (Mesiobuccal, Midbuccal, Distobuccal, Mesiolingual, Midlingual, Distolingual).

CLINICAL ATTACHMENT LEVEL (CAL) (Carranza):

Clinical Attachment Level was measured from the Cemento – Enamel Junction (CEJ) to the base of the pocket using William's Periodontal Probe.

- When the gingival margin was located on the anatomic crown, the level of the attachment was determined by subtracting from the probing pocket depth, the distance from the gingival margin to the CEJ. If both were the same, the loss of attachment was calculated to be zero.
- When the gingival margin coincided with the CEJ, the loss of attachment was calculated as equaling the probing pocket depth.
- When the gingival margin was located apical to the CEJ, the loss of attachment was greater than the probing pocket depth and therefore the distance between the CEJ and the gingival margin were added to the PPD.

Three measurements were made on the buccal aspect and three on the lingual aspect of each tooth – total of six sites per tooth (Mesiobuccal, Midbuccal, Distobuccal, Mesiolingual, Midlingual, and Distolingual).

ESTIMATION OF C-REACTIVE PROTEIN (CRP-hs):

LATEX HIGH SENSITIVITY :

PRINCIPLE OF THE METHOD:

Serum C-REACTIVE PROTEIN (CRP) causes agglutination of the latex particles coated with anti-human C-REACTIVE PROTEIN**.** This agglutination of the latex particles is proportional to the CRP concentration and was measured by turbidimetry.

CONTENTS AND COMPOSITION:

A. Reagent: 1 x 40 mL. Glycine buffer 0.1 mol/L, sodium azide 0.95 g/L, pH 8.6.

B. Reagent: 1 x 10 mL. Suspension of latex particles coated with anti-human CRP antibodies, sodium azide 0.95 g/L

S. CRP-hs Standard: For 1 x 5 mL Human serum. C-reactive protein concentration is stated on the vial label.

Human serum used in the preparation of the standard has been tested and found to be negative for the presence of antibodies anti-HIV and anti-HCV, as well as for HBs antigen However, the standard was handled cautiously.

STORAGE

Samples were stored at 2-8°C.

REAGENT PREPARATION

Working Reagent: Reagent B was mixed thoroughly with Reagent A in a vial. This mixture is stable for 20 days at **2-8°C.**

Smaller Working Reagent volumes was prepared by mixing: 1 ml of Reagent B + 4 mL of Reagent A. Reagent B vial was shaken well before pipetting.

CRP-hs Standard (S): Reconstituted with 5.0 mL of distilled water. Stable for 1 month at 2-8°C.

Calibration curve: Dilutions of the CRP-hs Standard was prepared using 9 g/L saline as diluent.

Concentration of the CRP-hs Standard was multiplied by the corresponding factor indicated below to obtain the CRP-hs concentration.

DILUTION	1	2	3	4	5
CRP-hs Standard(µl) Saline(µl)	10 70	20 60	40 40	60 20	80 -
Factor	0.125	0.25	0.5	0.75	1.0

ADDITIONAL EQUIPMENT:

Thermostatic water bath was set at 37°C.

Analyser or spectrophotometer or photometer thermostable at 37°Cwas used to read at 540 ± 20nm.

SAMPLES:

Serum collected by standard procedures. CRP in serum stable for 7 days at 2-8'C.

PROCEDURE:

1The working reagent and the instrument were brought to 37°C.

2. The instrument is zeroed with distilled water.

3. The mix was pipetted into a cuvette:

Working Reagent	1.5 ml
Water (Blank), Standard (S) or Sample	20 µl

4. The sample was mixed well and the cuvette is immediately inserted into the instrument.

5. Recording was done with the absorbance at 540 nm after 10 seconds (A_1) and after 5 minutes (A_2)

Calibration curve: The absorbance difference ($A_2 - A_1$) of each point was calculated and the values were plotted against CRP-hs concentration in a caliberation curve. CRP-hs concentration in the sample were calculated by interpolation of its absorbance (A_2-A_1) on the calibration curve.

REFERENCE VALUES:

Normal < 3mg/l
Increased >3mg/l

PURPOSE OF ESTIMATING BLOOD CHOLESTROL:

Quantitative estimation of Cholesterol in human serum is done by **Cholesterol oxidase - peroxidase/ end point method.** Cholesterol measurements are used in the diagnosis and treatment of hypercholesterolemias, atherosclerotic coronary artery disease. Cholesterol measurements are also used in the diagnosis of metabolic disorders involving lipids and lipoproteins. Total serum cholesterol concentrations depend on many factors including age, gender, diet, physical activity, liver disease, and other metabolic disorders.

PURPOSE OF ESTIMATING BLOOD TRIGLYCERIDES:

Quantitative estimation of Triglycerides in human serum by **GOP-Trinder End point method.** Triglyceride measurements are used in the diagnosis and treatment of patients with diabetes mellitus, nephrosis, liver obstruction, other diseases involving lipid metabolism, or various endocrine disorders**.**

PURPOSE OF ESTIMATING HDL:

Quantitative estimation of HDL CHOLESTEROL in human serum by **Direct Homogeneous End point method.** HDL cholesterol is inversely related to the risk of developing coronary artery disease. A low HDL/ LDL cholesterol ratio is directly related to the risk of developing coronary artery disease.

PURPOSE OF ESTIMATING LDL:

Quantitative estimation of LDL Cholesterol in human serum Direct enzymatic assay. Measurement of serum LDL cholesterol is useful in the screening of the lipid status of the individual to detect atherosclerotic risks and in monitoring the response to lipid lowering measures and also in the diagnosis and classification of hyperlipidemias.

In This Study Complete Lipid Profile Were Estimated To Assess Its Influence Over The Periodontal Status Of The Patients

PRINCIPLE OF ESTIMATING BLOOD CHOLESTROL:

CHOL reagent was used to measure cholesterol concentration by a timed-endpoint method .In the reaction cholesterol esterase (CE) hydrolyzes cholesterol esters to free cholesterol and fatty acids. Free cholesterol is oxidized to cholestene-3-one and

hydrogen peroxide by cholesterol oxidase (CO). Peroxidase catalyzes the reaction of hydrogen peroxide with 4-aminoantipyrine (4-AAP) and phenol to produce a colored quinoneimine product.

Cholesterol ester $\xrightarrow{CE}$ Cholesterol + Fatty Acid

Cholesterol + 0_2 $\xrightarrow{co}$ cholestene-3-one + H_2O_2

$2H_20_2$ + 4-AAP + Phenol $\xrightarrow{peroxidase}$ Quinoneimine + H_20

PRINCIPLE OF ESTIMATING BLOOD TRIGLYCERIDES:

GPO reagent was used to measure the triglycerides concentration by a timed endpoint method. Triglycerides in the sample are hydrolyzed to glycerol and free fatty acids by the action of lipase. A sequence of three coupled enzymatic steps using glycerol kinase (GK), glycerophosphate oxidase (GPO), and horseradish peroxidase (HPO) causes the oxidative coupling of 3,5-dichloro-2-hydroxybenzenesulfonic acid (DHBS) with 4-aminoantipyrine to form a red quinoneimine dye.

a) Triglycerides $\xrightarrow{Lipase}$ Glycerol + Fatty Acids

b) Glycerol + ATP $\xrightarrow[Mg]{GK}$ Glycerol-3-phosphate + ADP

(c) Glycerol-phosphate + 0_2 $\xrightarrow{GPO}$ Dihydroxyacetone + H_20_2

(d) $2H_20_2$ + 4-Aminoantipyrine + DHBS $\longrightarrow$ Quinoneimine Dye + HCl+ 2H 0

The SYNCHRON CX® System(s) automatically proportions the appropriate sample and reagent volumes into the cuvette. The ratio used was one part sample to 100 parts reagent. The system monitors the change in absorbance at 520 nanometers. This change in absorbance was directly proportional to the concentration of CHOL and TG in the

sample and was used by the System to calculate and express CHOL and TG concentration.

PRINCIPLE OF ESTIMATING HDL:

This direct HDL Cholesterol method is a homogeneous assay without the need for any offline pre-treatment or centrifugation steps. The method depends on a unique detergent which solubilizes only the HDL lipoprotein particles and releases HDL cholesterol to react with cholesterol esterase and cholesterol oxidase in the presence of chromogens, to produce a color product. The same detergent also inhibits the reaction of the cholesterol enzymes with LDL, VLDL, and chylomicrons lipoproteins by adsorbing to their surfaces. A polyanion contained in the reagent enhances the selectivity for HDL cholesterol assay by complexing LDL, VLDL, and chylomicrons lipoproteins. HDLD reagent is used to measure the cholesterol concentration by a timed-endpoint.

The SYNCTRON CX® System(s) automatically proportions the appropriate HDL cholesterol reagent and sample volumes into a cuvette. The ratio used was one part sample to 93 parts reagent. The System monitors the change in absorbance at 560 nanometers. This change in absorbance is directly proportional to the concentration of cholesterol in the sample and is used by the System to calculate and express the HDL-cholesterol concentration.

LDL, VLDL, Chylomicrons $\xrightarrow{\text{Detergent}}$ Stable Complexes

HDL Cholesterol $\xrightarrow{\text{Cholesterol Esterase}}$ A^4 Cholestenone + $H_?o_?$

$H_?o_?$ + DSBmT + 4-AA $\longrightarrow$ Color Product

DSBmT: NN-bis {4-sulphobytyl) - m – toluidine disodium, 4-AA: 4-aminoantipyrine

PRINCIPLE OF ESTIMATING LDL:

This direct LDL Cholesterol method is a homogeneous assay without the need for any offline pretreatment or centrifugation steps. The method depends on a unique detergent which solubilizes only the non-LDL lipoprotein particles and releases cholesterol to react with cholesterol esterase and cholesterol oxidase to produce a non-color forming reaction. A second detergent solubilizes the remaining LDL particles, and a chromogenic coupler allows for color formation. LDLD reagent is used to measure the cholesterol concentration by a timed-endpoint method.

The SYNCHRON CX System(s) automatically proportions the appropriate LDL cholesterol sample and reagent volumes into a cuvette. The ratio used is one part sample to 93 parts reagent. The System monitors the change in absorbance at 560 nanometers. This change in absorbance is directly proportional to the concentration of LDL cholesterol in the sample and is used by the System to calculate and express the LDL cholesterol concentration.

Detergent 1

HDL-C, VLDL-C,CM-C $\xrightarrow[\text{Cholesterol Oxidase}]{\text{Cholesterol Esterase}}$ Non-Color Product

Detergent 2

LDL Cholesterol $\xrightarrow[\text{Cholesterol Oxidase}]{\text{Cholesterol Esterase}}$ Δ^4 Cholestenone + H_2O_2

H_20_2 + DSBmT + 4-AA $\xrightarrow{\text{Perioxidase}}$ Color Product

C = Cholesterol
DSBmT = N,N-bis (4-sulphobutyl)-m-toluidine-disodium
4-AA = 4-aminoantipyrine

PERFORMANCE SIGNIFICANCE :

For cholesterol:

Linearity: This assay is linear for concentration up to 750 mg/dL.

Measurement Range: This method has a measurement range of 5 - 750 mg/dL

Sensitivity: The analytical sensitivity of this assay is 5 mg/dL.

For TG:

Linearity: This assay is linear for concentration up to 1000 mg/dL

Measurement Range: This method has a measurement range of 10 to 1000 mg/dL

Sensitivity: The analytical sensitivity of this assay is 10 mg/dL.

For HDL:

Linearity: This assay is linear for concentration up to 150 mg/dL.

Measurement Range: This method has a measurement range of 2to 150 mg/dL.

Sensitivity: The analytical sensitivity of this assay is 2 mg/dL.

For LDL:

Linearity: This assay is linear for concentration up to 450 mg/dL.

Measurement Range: This method has a measurement range of 1 to 450 mg/dL.

Sensitivity: The analytical sensitivity of this assay is l mg/dL.

Primary sampling:

1. Only serum was used as specimen for the test.

2. 1.5-2.5 ml of venous blood was collected from a peripheral vein in a yellow topped vacutainer tube. Serum was separated within 2 hours of collection.

3. The tube was allowed to stand at room temperature till complete clot formation occurs (for 30 minutes). It was ensured that complete clot formation has occurred and there are no fibrin threads.

4. All samples were processed on the same day within 2 hours of collection.

5. If assay was likely to be done after 48 hours of collection, the serum was stored at -20°C.

6. Hemolysed, contaminated samples were excluded from testing.

Type of Container and additive: yellow topped vacutainer tubes were used. No additive or anticoagulants were added

Calibration Procedure:

The system was calibrated or assayed using the Synchron CX MULTI Calibrator. The system has a valid calibration in memory before controls or patient samples were run.

The details were recorded in the calibration register.

The calibration report was used in conjunction with quality control results to determine the validity of calibration

Step by Step Procedure:

The reagent was loaded and then the system is calibrated as required. The reagent status was checked

The controls were checked before processing patient samples and then the sample were ensured to be at room temperature before loading.

The vacutainer was loaded with minimum volume of sample required.

Reference Interval/ Range:

CHOLESTEROL

Less than 200 mg/dL - low risk

201 to 239 mg/dL - borderline risk

240 mg/dL and greater - high risk

TGL

CONVENTIONAL UNITS

Normal	Less than 150 mg/dL
Borderline high	150-199 mg/d
High	200 - 500 mg
Very High	Greater than 500 mg/dL

HDL

Serum or Plasma (Male) : 27 - 67 mg/dL

Serum or Plasma (Female) : 29 - 89 mg/dL

LDL

Desirable : <130mg/dl

With high risk: >130 mg/dl

Potential source of variability:

Presence of fibrin in the sample may lead to erroneous results and hence centrifuging the sample was done only after complete clot formation.

Lysed and contaminated samples can lead to erroneously high values.

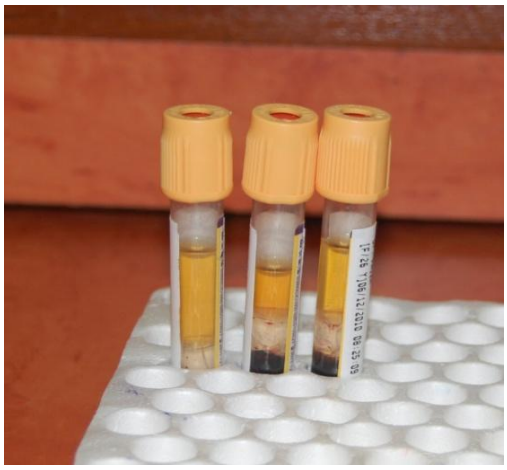
Reagent was thoroughly mixed and packed gently by inverting several times before loading on to the instrument.

Haemoglobin, bilirubin, lipemia, ascorbic acid, albumin and urea may affect the test results.

Photograph 1 : Armamentarium

Photograph 2: Williams's periodontal probe used to measure probing pocket depth and CAL

Photograph No. 3 Collection of blood sample



Photograph no. 4 samples & armamentarium for sample transportation

Photograph No. 5 SYNCHRON CX9 PRO CALIBRATOR

Photograph No. 6 SEMI- AUTOMATIC CALIBRATOR

STATISTICAL ANALYSIS

The statistical analysis was done using the computer software program SPSS version 12.

Mean and Standard Deviation were estimated for different variables in each study group.

In the present study, *P-value <0.05* **was considered as the level of significance**. **CHI SQUARE TEST FOR INDEPENDENCE OF ATTRIBUTES**

$$\chi^2 = \sum\left(\frac{(O-E)^2}{E}\right) \sim \chi^2_{(r-1)(c-1)\,\mathrm{df}}$$

$$\text{where } \mathrm{E} = \frac{(RT)(CT)}{\mathrm{GT}}$$

CORRELATION COEFFICIENT

$$r = \frac{\frac{\sum xy}{n} - \left(\frac{\sum x}{n}\right)\left(\frac{\sum y}{n}\right)}{\sqrt{\frac{\sum x^2}{n} - \left(\frac{\sum x}{n}\right)^2}\sqrt{\frac{\sum y^2}{n} - \left(\frac{\sum y}{n}\right)^2}}$$

Where O = Observed frequency in a cell

E = Expected frequency in a cell

RESULTS

A total of 100 patients in the age group of 18- 59 years including males and females were selected for the study. The study population was categorized into two groups based on their age; the younger age group comprising 50 subjects between the ages 18-34, and the middle aged group comprising 50 subjects between the ages 35-59(**Mohammad S. Al-Zahrani et al, 2003**)

Subjects within these two groups were further sub-divided into 2 subgroups depending on their body mass indices. One subgroup comprised of 25 subjects each. The BMI cut off decided to categorize the subjects was 22.9 **(Ministry of health, India, 2009)**. Similarly the 2nd group comprising of age range 35-59 years was also divided in the same way into 2 subgroups. Both males and females participated in the study irrespective of gender.

To summarize, the subjects were divided in the following ways

GROUP I: Age range from 18-34 years

Group I a: subjects having BMI 22.9 or less

Group I b: subjects having BMI 23 or more

GROUP II: Age range from 34-59 years

Group II a: subjects having BMI 22.9 or less

Group II b: subjects having BMI 23 or more

Table I shows the mean values of TC, TG, HDL, LDL, HSCRP,WH RATIO, BMI, CAL and LDL/HDL ratio among all the groups. The values are

150.40±22.66, 70.28 ± 30.90, 44.28 ± 11.66, 82.96 ± 25.92, 1.66 ± 1.86, 0.79 ± 0.05, 20.83 ± 1.71, 0.16 ± 0.41and 1.95 ± 0.69 respectively for group Ia,(table I)

164.96 ± 34.33, 97.84 ± 58.11, 36.13 ± 11.31, 77.67 ± 36.02, 5.36 ± 4.45, 0.9 ± 0.11, 30.97 ± 6.14, 1.29 ± 1.62 and 2.2 ± 0.99 respectively in group Ib, (table I)

184± 32.38, 101.16 ± 43.9, 42.31 ± 13.45, 101.52 ± 36.28, 2.18 ± 2.01, 0.83 ± 0.07, 22.26 ± 1.02, 2.10 ± 2.15, 2.57 ± 0.99 respectively in groupIIa (table I) and

194.36 ± 35.52, 122.71 ± 53.73, 43.49 ± 10.38, 110.64 ± 43.63, 8.74 ± 9.83, 0.89 ± 0.05, 33.43 ± 4.34, 4.05 ± 2.71, 2.60 ± 1.08 respectively in groupIIb(table I).

DISCUSSION

Obesity is known to affect host immunity **(Tanaka et al,1993, Stallone, 1994).** Upper body obesity (abdominal obesity) is believed to have greater ill-effects on health than lower body obesity **(Vanhala et al., 1997; Rexrode et al 1998).** The visceral fat accumulation that is frequently observed in upper body obesity increases the risk of cardiovascular diseases **(Nakamura et al., 1994)** and type 2 diabetes **(Banerji et al ,1995)**. An increase in visceral fat is associated with insulin resistance and increased liver fat **(Banerji et al, 1995; Goto et al 1995).**

Previous studies have showed a significant association between periodontitis and serum levels of AST, ALT, and cholinesterase suggesting that subjects with periodontitis also tend to have hepatic steatosis. Visceral fat, which leads to hepatic steatosis may also increase the risk of periodontitis.

Tumor necrosis factor α (TNF- α) mediates endotoxin induced injury in various organs and periodontal treatment **(Gemmel et al ,1997).** Among these organs, the liver is most involved in lipid metabolism. Adipose tissue also secretes TNF- α , which causes liver injury in obese rodents **(Yang et al ,1997).** Moreover TNF- α from adipose tissue was reported to be directly associated with insulin resistance **(Hotamisligil et al., 1996; Uysal et al., 1997).** An increase in hepatic triglyceride is dependent on an influx of free fatty acids, which are mainly derived from visceral adipose tissue, and is associated with insulin resistance **(Kopelman, 2000).** Several studies have indicated that hyperlipidemia frequently accompanies infectious diseases **(Feingold et al., 1992; Hardardottir al., 1994).** A single dose of bacterial endotoxin (LPS) can induce changes in lipid metabolism in adipose tissue and in the liver **(Feingold et al., 1992).**

Recently, an association between periodontitis and hyperlipidemia was reported **(Cutler et al., 1999).** If LPS derived from Gram-negative bacteria in periodontal pockets mediates TNF- α release from adipose tissue, it may be associated with hepatic dyslipidemia. Hepatic dyslipidemia induced by periodontal pathogens could be associated with insulin resistance.

In the present study both sexes were included similar to other studies (**Cutler 1999 , Joseph Katz 2002, Katz 2001 Loesche 2000).**

The sample selected in this study was within the age group of 18 to 34 years for younger age group and 35 to 59 years for middle age group was accordance with the study done by (**Al.Zaharani MS et al 2003)** .

The other confounding factors related to elevated cholesterol level like smoking, hypertension, lever disease were eliminated in the present study similar to earlier studies. (**Loesche 2000**).

According to **Katz et al 2002** smoking status is positively correlated with TC, LDL and TGL and negatively associated with HDL cholesterol. Vasoconstriction of gingival blood vessels caused by smoking promoted invasion of periodontal disease by microorganism (**Loesche 1998**), Hence smokers was excluded from the study.

Visceral fat accumulation raises the risk of cardiovascular disease more than does subcutaneous fat **(Nakamura et al., 1994).** Thereafter, a higher waist-hip ratio has been reported to increase this risk independently of BMI **(Rexrode et al., 1998).**

BMI is highly correlated with fat mass however, there are some limitations. For example, for the same BMI, older persons tend to have a higher body fat composition,

and, therefore, risk assessment by BMI is less accurate in older people (over 65 yrs of age). Furthermore, current BMI cut-off points for overweight and obesity are probably too high for Asian populations (Table 2) **(Choo, 2002).** More importantly, the BMI does not assess body fat distribution.

In this study both Body Mass Index and Central obesity were assessed as Clinical indicators of obesity. Blood lipid profile was estimated to assess the lipidaemic status of the subjects and levels of serum C reactive protein was estimated as systemic inflammatory marker associated with both obesity and periodontitis.

In group Ia, the average BMI was 20.83 (SD 1.71) and all the clinical and biochemical parameters were within the normal limits (Table I). In group IIb, the mean BMI was 30.97 (SD 6.14) which lies in the obesity range and the mean CAL was 1.29 (SD 1.62). Subjects in group IIb demonstrated increased CRP values of 5.36 (SD 4.45) (Table I).There was a positive correlation between CRP levels and the severity of periodontitis but the results were not statistically significant. In group IIa the mean BMI was 22.26(SD 1.02) and the mean CAL was 2.1(SD 2.15) which infers mild periodontitis was prevalent among the subjects and mean CRP level was 2.18±2.01 which was within the normal limits(Table I). In group IIb the mean BMI was33.43±4.34 and the mean CAL was 4.05±2.71.Even though there was a high degree of attachment loss and positive correlation the results were not statistically significant(Table I). This is in accordance with the studies done by **Saito et al, 2001** who concluded BMI was positively correlated with severity of periodontitis after adjustment with age and socioeconomic status. In the NHANES III portion of the study, BMI was positively related to the severity of

periodontal attachment loss. (**Robert J. Genco et al 2005).** Both smoking and obesity are independent risk indicators for periodontitis. (**Nishida et al 2005).**

Previous studies of the risk of cardiovascular disease with periodontitis adjusted the risk by using BMI as a conventional index of obesity **(Joshipura et al., 1996; Beck et al.,, 2000; Mattila et al., 2000).** The risk of cardiovascular disease with periodontitis should be reconsidered, since visceral fat accumulation appears to be related to both diseases and may be a confounding factor. The mean values of waist hip ratio among males is 0.83 ± 0.03, 0.97 ± 0.02, 0.87 ± 0.03 and 0.81 in groups Ia, Ib, IIa and IIb respectively while for the females 0.75 ± 0.03, 0.88 ± 0.11, 0.77 ± 0.06, 0.89 ± 0.05 in groups Ia, Ib, IIa and IIb respectively (Table XIX). In group IIa the waist hip ratio had significant positive correlation with CAL without gender predilection ($p < 0.05$) (Table no II). There was similar positive correlation in group IIb as well without gender predilection ($p < 0.05$). There was no statistical significance between waist hip ratio and CAL levels in group Ia and Ib categories(Table II). This infers that the CAL is mainly age related and central obesity could be an additional factor when combined with advancing age for periodontitis.

Previous studies have indicated that patients with adult periodontitis often have elevated serum triglyceride and LDL levels. (**Cutler et al 1990).** Results of studies done by Cutler et al has proved that significant positive relationship exists between periodontitis and elevated serum levels of triglycerides and IgG antibodies against P.gingivalis LPS. Moreover TG can increase the amount of IL-1β released by PMN's in response to P.gingivalis LPS. (**Cutler, Edward et al 1999).**

The mean values of TC in the present study are 150.40±22.6, 164.96±34.33, 184.32±32.38 and 194.36±35.52 among group Ia, Ib, IIa and IIb respectively. (Table I)

The mean values of TG in the present study are 70.28 ± 30.90, 97.84±58.11, 101.16±43.90 and 122.71±53.73 among group Ia, Ib, IIa and IIb respectively. (Table I)

The mean values of HDL in the present study are 44.28 ± 11.66, 36.13±11.31, 42.31±13.45 and 43.49±10.38 among group Ia, Ib, IIa and IIb respectively. (Table I)

The mean values of LDL in the present study are 82.36 ± 25.92, 77.67±36.02, 101.52±36.28 and 110.64±43.63 among group Ia, Ib, IIa and IIb respectively. (Table I)

The mean values of LDL/HDL ratio in the present study are 1.95 ± 0.69, 2.20±0.99, 2.57±0.99 and 2.60±1.08 among group Ia, Ib, IIa and IIb respectively. (Table I)

In the present study the mean lipid profile had no statistically significant correlation with the CAL in all the groups. The mean values of the lipid profile in the present study are within the normal limits and hence could be a possible reason for not achieving statistically significant positive correlation with CAL. (Table II)

On the contrary studies have indicated that increased serum TG has a negative influence on all the clinical measures of periodontal health particularly in patients without preexisting periodontitis. (**Cutler . W, Robert .L et al 1999).**

Total cholesterol, LDL and TG were significantly higher in periodontally diseased subjects compared to controls. (**Losche et al 2000).**

Studies conducted by **Joseph Katz et al 2002** has concluded that the presence of periodontal pockets as measured by CPITN was positively associated with total cholesterol and LDL.

The mean values of hsCRP in the present study are 1.66 ± 1.86, 5.36±4.45, 2.18±2.01 and 8.74±9.83 among group Ia, Ib, IIa and IIb respectively. (Table I) The hsCRP levels are highly positively statistically significant correlation with the CAL measurement in group Ib subjects. (Table II) This may infer that increased CRP levels in younger age group subjects with high BMI could be a possible risk indicator for increased attachment loss. Hence it may be hypothesized here that increased BMI at an younger age group can pose an increased risk for periodontitis and attachment loss.

This study is in agreement with **Al-Zahrani et al 2003** who stated that in younger population overall and abdominal obesity are associated with increased prevalence of periodontal disease.

Numerous studies have shown a positive association between chronic periodontitis and elevated CRP levels (**Ebersole et al, 1997, Ide et al 2004, D,Aiuto 2005, Pussinen et al 2005).**

According to **American Journal of Cardiology & Journal of Periodontology, 2009**, patients with periodontitis meeting criteria for metabolic syndrome should be identified and all the risk factors for atherosclerotic CVD should be treated, beginning with lifestyle changes aimed at weight reduction.

Finally, although longitudinal evaluation with a larger population may be an important step in revealing the causal relationship between periodontal disease and obesity, impaired lipid metabolism our study may shed light on the clarification of this association in the future.

Tables I: Mean values of the parameters assessed in the study

	Group							
	Group IA		Group IB		Group 2A		Group 2B	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
TC	150.40	22.66	164.96	34.33	184.32	32.38	194.36	35.52
TGL	70.28	30.90	97.84	58.11	101.16	43.90	122.71	53.73
HDL	44.28	11.66	36.13	11.31	42.31	13.45	43.49	10.38
LDL	82.96	25.92	77.67	36.02	101.52	36.28	110.64	43.63
HSCRP	1.66	1.86	5.36	4.45	2.18	2.01	8.74	9.83
WHRatio	.79	.05	.90	.11	.83	.07	.89	.05
BMI_A	20.83	1.71	30.97	6.14	22.26	1.02	33.43	4.34
PD	2.21	.54	3.36	1.11	3.74	1.74	4.77	1.52
CAL	.16	.41	1.29	1.62	2.10	2.15	4.05	2.71
BI	20.70	21.72	63.20	30.65	58.08	31.61	84.76	24.31
PI	.48	.54	1.22	.73	1.46	.85	1.96	.87
LDL / HDL	1.95	.69	2.20	.99	2.57	.99	2.60	1.08

Table II Association between biochemical, clinical and periodontal parameters in group Ia

		PD	CAL	BI
TC	Pearson Correlation	-.345	-.363	-.449(*)
	Sig. (2-tailed)	.091	.074	.024
	N	25	25	25
TGL	Pearson Correlation	.165	.155	.021
	Sig. (2-tailed)	.430	.460	.919
	N	25	25	25
HDL	Pearson Correlation	-.232	-.249	-.224
	Sig. (2-tailed)	.264	.229	.281
	N	25	25	25
LDL	Pearson Correlation	-.366	-.318	-.417(*)
	Sig. (2-tailed)	.072	.122	.038
	N	25	25	25
HSCRP	Pearson Correlation	.018	.131	.022
	Sig. (2-tailed)	.932	.532	.915
	N	25	25	25
WHRatio	Pearson Correlation	-.037	.260	.057
	Sig. (2-tailed)	.862	.210	.787
	N	25	25	25
BMI_A	Pearson Correlation	.036	.354	.090
	Sig. (2-tailed)	.866	.083	.667
	N	25	25	25
LDL / HDL	Pearson Correlation	-.178	-.103	-.244
	Sig. (2-tailed)	.394	.624	.240
	N	25	25	25

TableIII : ASSOCIATION BETWEEN BIOCHEMICAL, CLINICAL AND PERIODONTAL PARAMETERS IN GROUP IB

		PD	CAL	BI
TC	Pearson Correlation	-.407(*)	-.336	-.226
	Sig. (2-tailed)	.044	.101	.278
	N	25	25	25
TGL	Pearson Correlation	.058	.155	.033
	Sig. (2-tailed)	.782	.458	.876
	N	25	25	25
HDL	Pearson Correlation	-.347	-.169	-.422(*)
	Sig. (2-tailed)	.089	.418	.036
	N	25	25	25
LDL	Pearson Correlation	-.230	-.024	-.137
	Sig. (2-tailed)	.269	.911	.515
	N	25	25	25
HSCRP	Pearson Correlation	.485(*)	.524(**)	.347
	Sig. (2-tailed)	.014	.007	.089
	N	25	25	25
WHRatio	Pearson Correlation	.101	.100	.323
	Sig. (2-tailed)	.631	.634	.115
	N	25	25	25
BMI_A	Pearson Correlation	.139	.066	.417(*)
	Sig. (2-tailed)	.509	.753	.038
	N	25	25	25
LDL / HDL	Pearson Correlation	-.002	.089	.192
	Sig. (2-tailed)	.993	.674	.357
	N	25	25	25

Table IV : Association Between Biochemical, Clinical And Periodontal Parameters In Group IIa

		PD	CAL	BI
TC	Pearson Correlation	-.131	-.092	-.226
	Sig. (2-tailed)	.533	.663	.278
	N	25	25	25
TGL	Pearson Correlation	.078	.112	.014
	Sig. (2-tailed)	.712	.593	.945
	N	25	25	25
HDL	Pearson Correlation	.002	-.021	-.169
	Sig. (2-tailed)	.993	.920	.418
	N	25	25	25
LDL	Pearson Correlation	-.156	-.090	-.310
	Sig. (2-tailed)	.456	.667	.132
	N	25	25	25
HSCRP	Pearson Correlation	.167	.104	.327
	Sig. (2-tailed)	.424	.620	.111
	N	25	25	25
WHRatio	Pearson Correlation	.392	.408(*)	.374
	Sig. (2-tailed)	.053	.043	.066
	N	25	25	25
BMI_A	Pearson Correlation	.154	.146	.161
	Sig. (2-tailed)	.463	.486	.441
	N	25	25	25
LDL / HDL	Pearson Correlation	-.248	-.153	-.240
	Sig. (2-tailed)	.233	.464	.249
	N	25	25	25

* Correlation is significant at the 0.05 level (2-tailed).

Table V : Association Between Biochemical, Clinical And Periodontal Parameters In Group IIb

		PD	CAL	BI
TC	Pearson Correlation	.111	-.075	.324
	Sig. (2-tailed)	.596	.723	.114
	N	25	25	25
TGL	Pearson Correlation	-.226	-.148	.259
	Sig. (2-tailed)	.277	.480	.211
	N	25	25	25
HDL	Pearson Correlation	.257	.103	.221
	Sig. (2-tailed)	.215	.625	.288
	N	25	25	25
LDL	Pearson Correlation	.109	.017	.227
	Sig. (2-tailed)	.603	.934	.276
	N	25	25	25
HSCRP	Pearson Correlation	-.010	.392	.265
	Sig. (2-tailed)	.963	.053	.201
	N	25	25	25
WHRatio	Pearson Correlation	.294	.417(*)	.003
	Sig. (2-tailed)	.154	.038	.990
	N	25	25	25
BMI_A	Pearson Correlation	.194	.040	.077
	Sig. (2-tailed)	.353	.848	.714
	N	25	25	25
LDL / HDL	Pearson Correlation	-.148	-.138	-.016
	Sig. (2-tailed)	.479	.511	.938
	N	25	25	25

TABLE VI:Distribution Of Severity Of Periodontitis Among The Study Groups:

			CAL Group				
			0-1	1-3	3-5	Above 5	Total
Group	Group IA	Count	23	2	0	0	25
		% within Group	92.0%	8.0%	.0%	.0%	100.0%
		% within CAL Group	44.2%	10.5%	.0%	.0%	25.0%
	Group IB	Count	16	4	4	1	25
		% within Group	64.0%	16.0%	16.0%	4.0%	100.0%
		% within CAL Group	30.8%	21.1%	28.6%	6.7%	25.0%
	Group 2A	Count	9	8	4	4	25
		% within Group	36.0%	32.0%	16.0%	16.0%	100.0%
		% within CAL Group	17.3%	42.1%	28.6%	26.7%	25.0%
	Group 2B	Count	4	5	6	10	25
		% within Group	16.0%	20.0%	24.0%	40.0%	100.0%
		% within CAL Group	7.7%	26.3%	42.9%	66.7%	25.0%
Total		Count	52	19	14	15	100
		% within Group	52.0%	19.0%	14.0%	15.0%	100.0%
		% within CAL Group	100.0%	100.0%	100.0%	100.0%	100.0%

TABLE VII: Prevalence of periodontitis with respect to TC levels:

Group				CAL Group				Total
				0-1	1-3	3-5	Above 5	
Group IA	TC - Group	Below 200	Count	23	2			25
			% within TC -	92.0%	8.0%			100.0%
			% within CAL Group	100.0%	100.0%			100.0%
	Total		Count	23	2			25
			% within TC - Group	92.0%	8.0%			100.0%
			% within CAL Group	100.0%	100.0%			100.0%
Group IB	TC - Group	Below 200	Count	12	3	4	1	20
			% within TC - Group	60.0%	15.0%	20.0%	5.0%	100.0%
			% within CAL Group	75.0%	75.0%	100.0%	100.0%	80.0%
		Above 200	Count	4	1	0	0	5
			% within TC - Group	80.0%	20.0%	.0%	.0%	100.0%
			% within CAL Group	25.0%	25.0%	.0%	.0%	20.0%
	Total		Count	16	4	4	1	25
			% within TC - Group	64.0%	16.0%	16.0%	4.0%	100.0%
			% within CAL Group	100.0%	100.0%	100.0%	100.0%	100.0%
Group 2A	TC - Group	Below 200	Count	6	6	2	4	18
			% within TC - Group	33.3%	33.3%	11.1%	22.2%	100.0%
			% within CAL Group	66.7%	75.0%	50.0%	100.0%	72.0%
		Above 200	Count	3	2	2	0	7
			% within TC - Group	42.9%	28.6%	28.6%	.0%	100.0%
			% within CAL Group	33.3%	25.0%	50.0%	.0%	28.0%
	Total		Count	9	8	4	4	25
			% within TC - Group	36.0%	32.0%	16.0%	16.0%	100.0%
			% within CAL Group	100.0%	100.0%	100.0%	100.0%	100.0%
Group 2B	TC - Group	Below 200	Count	2	4	5	7	18
			% within TC - Group	11.1%	22.2%	27.8%	38.9%	100.0%
			% within CAL Group	50.0%	80.0%	83.3%	70.0%	72.0%
		Above 200	Count	2	1	1	3	7
			% within TC - Group	28.6%	14.3%	14.3%	42.9%	100.0%
			% within CAL Group	50.0%	20.0%	16.7%	30.0%	28.0%
	Total		Count	4	5	6	10	25
			% within TC - Group	16.0%	20.0%	24.0%	40.0%	100.0%
			% within CAL Group	100.0%	100.0%	100.0%	100.0%	100.0%

TABLE VIII: CORRELATION BETWEEN TC AND CAL

Group		Value	df	Asymp. Sig. (2-sided)
Group IA	Pearson Chi-Square	.(a)		
	N of Valid Cases	25		
Group IB	Pearson Chi-Square	1.562(b)	3	.668
	Likelihood Ratio	2.527	3	.470
	Linear-by-Linear Association	1.200	1	.273
	N of Valid Cases	25		
Group 2A	Pearson Chi-Square	2.679(c)	3	.444
	Likelihood Ratio	3.648	3	.302
	Linear-by-Linear Association	.563	1	.453
	N of Valid Cases	25		
Group 2B	Pearson Chi-Square	1.521(d)	3	.677
	Likelihood Ratio	1.474	3	.688
	Linear-by-Linear Association	.209	1	.647
	N of Valid Cases	25		

TABLE IX: Prevalence of periodontitis with respect to LDL levels:

Group				CAL Group				Total
				0-1	1-3	3-5	Above 5	
Group IA	LDL - Group	Below 130	Count	22	2			24
			% within LDL - Group	91.7%	8.3%			100.0%
			% within CAL Group	95.7%	100.0%			96.0%
		Above 130	Count	1	0			1
			% within LDL - Group	100.0%	.0%			100.0%
			% within CAL Group	4.3%	.0%			4.0%
	Total		**Count**	**23**	**2**			**25**
			% within LDL - Group	**92.0%**	**8.0%**			**100.0%**
			% within CAL Group	**100.0%**	**100.0%**			**100.0%**
ssGroup IB	**LDL - Group**	**Below 130**	**Count**	**15**	**4**	**4**	**1**	**24**
			% within LDL - Group	**62.5%**	**16.7%**	**16.7%**	**4.2%**	**100.0%**
			% within CAL Group	**93.8%**	**100.0%**	**100.0%**	**100.0%**	**96.0%**
		Above 130	**Count**	**1**	**0**	**0**	**0**	**1**
			% within LDL - Group	**100.0%**	**.0%**	**.0%**	**.0%**	**100.0%**
			% within CAL Group	**6.3%**	**.0%**	**.0%**	**.0%**	**4.0%**
	Total		**Count**	**16**	**4**	**4**	**1**	**25**
			% within LDL - Group	**64.0%**	**16.0%**	**16.0%**	**4.0%**	**100.0%**
			% within CAL Group	**100.0%**	**100.0%**	**100.0%**	**100.0%**	**100.0%**
Group 2A	**LDL - Group**	**Below 130**	**Count**	**6**	**6**	**3**	**4**	**19**
			% within LDL - Group	**31.6%**	**31.6%**	**15.8%**	**21.1%**	**100.0%**
			% within CAL Group	**66.7%**	**75.0%**	**75.0%**	**100.0%**	**76.0%**
		Above 130	**Count**	**3**	**2**	**1**	**0**	**6**
			% within LDL - Group	**50.0%**	**33.3%**	**16.7%**	**.0%**	**100.0%**
			% within CAL Group	**33.3%**	**25.0%**	**25.0%**	**.0%**	**24.0%**
	Total		**Count**	**9**	**8**	**4**	**4**	**25**
			% within LDL - Group	**36.0%**	**32.0%**	**16.0%**	**16.0%**	**100.0%**
			% within CAL Group	**100.0%**	**100.0%**	**100.0%**	**100.0%**	**100.0%**
Group 2B	**LDL - Group**	**Below 130**	**Count**	**2**	**3**	**5**	**6**	**16**
			% within LDL - Group	12.5%	18.8%	31.3%	37.5%	100.0%
			% within CAL Group	50.0%	60.0%	83.3%	60.0%	64.0%
		Above 130	Count	2	2	1	4	9
			% within LDL - Group	22.2%	22.2%	11.1%	44.4%	100.0%
			% within CAL Group	50.0%	40.0%	16.7%	40.0%	36.0%
	Total		Count	4	5	6	10	25
			% within LDL - Group	16.0%	20.0%	24.0%	40.0%	100.0%
			% within CAL Group	100.0%	100.0%	100.0%	100.0%	100.0%

TABLE X: CORRELATION BETWEEN LDL AND CAL

Group		Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Group IA	Pearson Chi-Square	.091(b)	1	.763		
	Continuity Correction(a)	.000	1	1.000		
	Likelihood Ratio	.170	1	.680		
	Fisher's Exact Test				1.000	.920
	Linear-by-Linear Association	.087	1	.768		
	N of Valid Cases	25				
Group IB	Pearson Chi-Square	.586(c)	3	.900		
	Likelihood Ratio	.916	3	.822		
	Linear-by-Linear Association	.450	1	.502		
	N of Valid Cases	25				
Group 2A	Pearson Chi-Square	1.700(d)	3	.637		
	Likelihood Ratio	2.601	3	.457		
	Linear-by-Linear Association	1.360	1	.244		
	N of Valid Cases	25				
Group 2B	Pearson Chi-Square	1.418(e)	3	.701		
	Likelihood Ratio	1.529	3	.676		
	Linear-by-Linear Association	.115	1	.734		
	N of Valid Cases	25				

TABLE XI: Prevalence of periodontitis with respect to HDL levels:

Group				CAL Group				Total
				0-1	1-3	3-5	Above 5	
Group IA	HDL - Group	Below 27	Count	1	0			1
			% within HDL - Group	100.0%	.0%			100.0%
			% within CAL Group	4.3%	.0%			4.0%
		Above 27	Count	22	2			24
			% within HDL - Group	91.7%	8.3%			100.0%
			% within CAL Group	95.7%	100.0%			96.0%
	Total		Count	23	2			25
			% within HDL - Group	92.0%	8.0%			100.0%
			% within CAL Group	100.0%	100.0%			100.0%
Group IB	HDL - Group	Below 27	Count	3	1	0	0	4
			% within HDL - Group	75.0%	25.0%	.0%	.0%	100.0%
			% within CAL Group	18.8%	25.0%	.0%	.0%	16.0%
		Above 27	Count	13	3	4	1	21
			% within HDL - Group	61.9%	14.3%	19.0%	4.8%	100.0%
			% within CAL Group	81.3%	75.0%	100.0%	100.0%	84.0%
	Total		Count	16	4	4	1	25
			% within HDL - Group	64.0%	16.0%	16.0%	4.0%	100.0%
			% within CAL Group	100.0%	100.0%	100.0%	100.0%	100.0%
Group 2A	HDL - Group	Below 27	Count	0	1	0	0	1
			% within HDL - Group	.0%	100.0%	.0%	.0%	100.0%
			% within CAL Group	.0%	12.5%	.0%	.0%	4.0%
		Above 27	Count	9	7	4	4	24
			% within HDL - Group	37.5%	29.2%	16.7%	16.7%	100.0%
			% within CAL Group	100.0%	87.5%	100.0%	100.0%	96.0%
	Total		Count	9	8	4	4	25
			% within HDL - Group	36.0%	32.0%	16.0%	16.0%	100.0%
			% within CAL Group	100.0%	100.0%	100.0%	100.0%	100.0%
Group 2B	HDL - Group	Below 27	Count	1	0	0	0	1
			% within HDL - Group	100.0%	.0%	.0%	.0%	100.0%
			% within CAL Group	25.0%	.0%	.0%	.0%	4.0%
		Above 27	Count	3	5	6	10	24
			% within HDL - Group	12.5%	20.8%	25.0%	41.7%	100.0%
			% within CAL Group	75.0%	100.0%	100.0%	100.0%	96.0%
	Total		Count	4	5	6	10	25
			% within HDL - Group	16.0%	20.0%	24.0%	40.0%	100.0%
			% within CAL Group	100.0%	100.0%	100.0%	100.0%	100.0%

TABLE XII : CORRELATION BETWEEN HDL AND CAL

Group		Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Group IA	Pearson Chi-Square	.091(b)	1	.763		
	Continuity Correction(a)	.000	1	1.000		
	Likelihood Ratio	.170	1	.680		
	Fisher's Exact Test				1.000	.920
	Linear-by-Linear Association	.087	1	.768		
	N of Valid Cases	25				
Group IB	Pearson Chi-Square	1.283(c)	3	.733		
	Likelihood Ratio	2.042	3	.564		
	Linear-by-Linear Association	.700	1	.403		
	N of Valid Cases	25				
Group 2A	Pearson Chi-Square	2.214(d)	3	.529		
	Likelihood Ratio	2.369	3	.499		
	Linear-by-Linear Association	.013	1	.911		
	N of Valid Cases	25				
Group 2B	Pearson Chi-Square	5.469(d)	3	.141		
	Likelihood Ratio	3.899	3	.273		
	Linear-by-Linear Association	2.884	1	.089		
	N of Valid Cases	25				

TABLE XIII: Prevalence of periodontitis with respect to TGL levels

Group				CAL Group				Total
				0-1	1-3	3-5	Above 5	
Group IA	TGL – Group	Below 150	Count	23	1			24
			% within TGL - Group	95.8%	4.2%			100.0%
			% within CAL Group	100.0%	50.0%			96.0%
		Above 150	Count	0	1			1
			% within TGL - Group	.0%	100.0%			100.0%
			% within CAL Group	.0%	50.0%			4.0%
	Total		Count	23	2			25
			% within TGL - Group	92.0%	8.0%			100.0%
			% within CAL Group	100.0%	100.0%			100.0%
Group IB	TGL - Group	Below 150	Count	13	4	3	1	21
			% within TGL - Group	61.9%	19.0%	14.3%	4.8%	100.0%
			% within CAL Group	81.3%	100.0%	75.0%	100.0%	84.0%
		Above 150	Count	3	0	1	0	4
			% within TGL - Group	75.0%	.0%	25.0%	.0%	100.0%
			% within CAL Group	18.8%	.0%	25.0%	.0%	16.0%
	Total		Count	16	4	4	1	25
			% within TGL - Group	64.0%	16.0%	16.0%	4.0%	100.0%
			% within CAL Group	100.0%	100.0%	100.0%	100.0%	100.0%
Group 2A	TGL - Group	Below 150	Count	7	7	4	3	21
			% within TGL - Group	33.3%	33.3%	19.0%	14.3%	100.0%
			% within CAL Group	77.8%	87.5%	100.0%	75.0%	84.0%
		Above 150	Count	2	1	0	1	4
			% within TGL - Group	50.0%	25.0%	.0%	25.0%	100.0%
			% within CAL Group	22.2%	12.5%	.0%	25.0%	16.0%
	Total		Count	9	8	4	4	25
			% within TGL - Group	36.0%	32.0%	16.0%	16.0%	100.0%
			% within CAL Group	100.0%	100.0%	100.0%	100.0%	100.0%
Group 2B	TGL - Group	Below 150	Count	4	3	3	7	17
			% within TGL - Group	23.5%	17.6%	17.6%	41.2%	100.0%
			% within CAL Group	100.0%	60.0%	50.0%	70.0%	68.0%
		Above 150	Count	0	2	3	3	8
			% within TGL - Group	.0%	25.0%	37.5%	37.5%	100.0%
			% within CAL Group	.0%	40.0%	50.0%	30.0%	32.0%
	Total		Count	4	5	6	10	25
			% within TGL - Group	16.0%	20.0%	24.0%	40.0%	100.0%
			% within CAL Group	100.0%	100.0%	100.0%	100.0%	100.0%

TABLE XIV: CORRELATION BETWEEN TGL AND CAL

Group		Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Group IA	Pearson Chi-Square	11.979(b)	1	.001		
	Continuity Correction(a)	2.497	1	.114		
	Likelihood Ratio	5.625	1	.018		
	Fisher's Exact Test				.080	.080
	Linear-by-Linear Association	11.500	1	.001		
	N of Valid Cases	25				
Group IB	Pearson Chi-Square	1.283(c)	3	.733		
	Likelihood Ratio	2.042	3	.564		
	Linear-by-Linear Association	.057	1	.811		
	N of Valid Cases	25				
Group 2A	Pearson Chi-Square	1.335(d)	3	.721		
	Likelihood Ratio	1.922	3	.589		
	Linear-by-Linear Association	.057	1	.811		
	N of Valid Cases	25				
Group 2B	Pearson Chi-Square	2.941(e)	3	.401		
	Likelihood Ratio	4.078	3	.253		
	Linear-by-Linear Association	.553	1	.457		
	N of Valid Cases	25				

TABLE XV: Prevalence of periodontitis with respect to HDL/LDL ratio levels

Group				CAL Group				Total
				0-1	1-3	3-5	Above 5	
Group IA	LDL / HDL	Below 3.5	Count	23	2			25
			% within LDL / HDL	92.0%	8.0%			100.0%
			% within CAL Group	100.0%	100.0%			100.0%
	Total		Count	23	2			25
			% within LDL / HDL	92.0%	8.0%			100.0%
			% within CAL Group	100.0%	100.0%			100.0%
Group IB	LDL / HDL	Below 3.5	Count	15	3	3	1	22
			% within LDL / HDL	68.2%	13.6%	13.6%	4.5%	100.0%
			% within CAL Group	93.8%	75.0%	75.0%	100.0%	88.0%
		Above 3.5	Count	1	1	1	0	3
			% within LDL / HDL	33.3%	33.3%	33.3%	.0%	100.0%
			% within CAL Group	6.3%	25.0%	25.0%	.0%	12.0%
	Total		Count	16	4	4	1	25
			% within LDL / HDL	64.0%	16.0%	16.0%	4.0%	100.0%
			% within CAL Group	100.0%	100.0%	100.0%	100.0%	100.0%
Group 2A	LDL / HDL	Below 3.5	Count	7	7	4	3	21
			% within LDL / HDL	33.3%	33.3%	19.0%	14.3%	100.0%
			% within CAL Group	77.8%	87.5%	100.0%	75.0%	84.0%
		Above 3.5	Count	2	1	0	1	4
			% within LDL / HDL	50.0%	25.0%	.0%	25.0%	100.0%
			% within CAL Group	22.2%	12.5%	.0%	25.0%	16.0%
	Total		Count	9	8	4	4	25
			% within LDL / HDL	36.0%	32.0%	16.0%	16.0%	100.0%
			% within CAL Group	100.0%	100.0%	100.0%	100.0%	100.0%
Group 2B	LDL / HDL	Below 3.5	Count	2	4	5	9	20
			% within LDL / HDL	10.0%	20.0%	25.0%	45.0%	100.0%
			% within CAL Group	50.0%	80.0%	83.3%	90.0%	80.0%
		Above 3.5	Count	2	1	1	1	5
			% within LDL / HDL	40.0%	20.0%	20.0%	20.0%	100.0%
			% within CAL Group	50.0%	20.0%	16.7%	10.0%	20.0%
	Total		Count	4	5	6	10	25
			% within LDL / HDL	16.0%	20.0%	24.0%	40.0%	100.0%
			% within CAL Group	100.0%	100.0%	100.0%	100.0%	100.0%

TABLE XVI: CORRELATION BETWEEN LDL/HDL AND CAL

Group		Value	df	Asymp. Sig. (2-sided)
Group IA	Pearson Chi-Square	.(a)		
	N of Valid Cases	25		
Group IB	Pearson Chi-Square	1.918(b)	3	.590
	Likelihood Ratio	1.868	3	.600
	Linear-by-Linear Association	.655	1	.418
	N of Valid Cases	25		
Group 2A	Pearson Chi-Square	1.335(c)	3	.721
	Likelihood Ratio	1.922	3	.589
	Linear-by-Linear Association	.057	1	.811
	N of Valid Cases	25		
Group 2B	Pearson Chi-Square	2.917(d)	3	.405
	Likelihood Ratio	2.563	3	.464
	Linear-by-Linear Association	2.264	1	.132
	N of Valid Cases	25		

TABLE XVII: Prevalence of periodontitis with respect to hscrp levels

Group				CAL Group				Total
				0-1	1-3	3-5	Above 5	
Group IA	HSCRP	Below 3	Count	21	1			22
			% within HSCRP	95.5%	4.5%			100.0%
			% within CAL Group	91.3%	50.0%			88.0%
		3-10	Count	2	1			3
			% within HSCRP	66.7%	33.3%			100.0%
			% within CAL Group	8.7%	50.0%			12.0%
	Total		Count	23	2			25
			% within HSCRP	92.0%	8.0%			100.0%
			% within CAL Group	100.0%	100.0%			100.0%
Group IB	HSCRP	Below 3	Count	8	1	1	0	10
			% within HSCRP	80.0%	10.0%	10.0%	.0%	100.0%
			% within CAL Group	50.0%	25.0%	25.0%	.0%	40.0%
		3-10	Count	6	2	1	0	9
			% within HSCRP	66.7%	22.2%	11.1%	.0%	100.0%
			% within CAL Group	37.5%	50.0%	25.0%	.0%	36.0%
		Above 10	Count	2	1	2	1	6
			% within HSCRP	33.3%	16.7%	33.3%	16.7%	100.0%
			% within CAL Group	12.5%	25.0%	50.0%	100.0%	24.0%
	Total		Count	16	4	4	1	25
			% within HSCRP	64.0%	16.0%	16.0%	4.0%	100.0%
			% within CAL Group	100.0%	100.0%	100.0%	100.0%	100.0%
Group 2A	HSCRP	Below 3	Count	9	4	4	2	19
			% within HSCRP	47.4%	21.1%	21.1%	10.5%	100.0%
			% within CAL Group	100.0%	50.0%	100.0%	50.0%	76.0%
		3-10	Count	0	4	0	2	6
			% within HSCRP	.0%	66.7%	.0%	33.3%	100.0%
			% within CAL Group	.0%	50.0%	.0%	50.0%	24.0%
	Total		Count	9	8	4	4	25
			% within HSCRP	36.0%	32.0%	16.0%	16.0%	100.0%
			% within CAL Group	100.0%	100.0%	100.0%	100.0%	100.0%
Group 2B	HSCRP	Below 3	Count	1	2	2	0	5
			% within HSCRP	20.0%	40.0%	40.0%	.0%	100.0%
			% within CAL Group	25.0%	40.0%	33.3%	.0%	20.0%
		3-10	Count	3	1	4	4	12
			% within HSCRP	25.0%	8.3%	33.3%	33.3%	100.0%
			% within CAL Group	75.0%	20.0%	66.7%	40.0%	48.0%
		Above 10	Count	0	2	0	6	8
			% within HSCRP	.0%	25.0%	.0%	75.0%	100.0%
			% within CAL Group	.0%	40.0%	.0%	60.0%	32.0%
	Total		Count	4	5	6	10	25
			% within HSCRP	16.0%	20.0%	24.0%	40.0%	100.0%
			% within CAL Group	100.0%	100.0%	100.0%	100.0%	100.0%

TABLE XVIII: CORRELATION BETWEEN hsCRP AND CAL

Group		Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Group IA	Pearson Chi-Square	2.973(b)	1	.085		
	Continuity Correction(a)	.348	1	.555		
	Likelihood Ratio	1.983	1	.159		
	Fisher's Exact Test				.230	.230
	Linear-by-Linear Association	2.854	1	.091		
	N of Valid Cases	25				
Group IB	Pearson Chi-Square	6.389(c)	6	.381		
	Likelihood Ratio	6.027	6	.420		
	Linear-by-Linear Association	4.278	1	.039		
	N of Valid Cases	25				
Group 2A	Pearson Chi-Square	8.553(d)	3	.036		
	Likelihood Ratio	10.918	3	.012		
	Linear-by-Linear Association	1.977	1	.160		
	N of Valid Cases	25				
Group 2B	Pearson Chi-Square	11.326(e)	6	.079		
	Likelihood Ratio	15.794	6	.015		
	Linear-by-Linear Association	4.331	1	.037		
	N of Valid Cases	25				

TABLE XIX: WHRatio

		Gender			
		Male		Female	
		Mean	SD	Mean	SD
Group	Group IA	.83	.03	.75	.03
	Group IB	.97	.02	.88	.11
	Group 2A	.87	.03	.77	.06
	Group 2B	.89	.	.89	.05

FIGURE I: COMPARISION BETWEEN TC, TG ,HDL AND LDL IN 4 GROUPS:

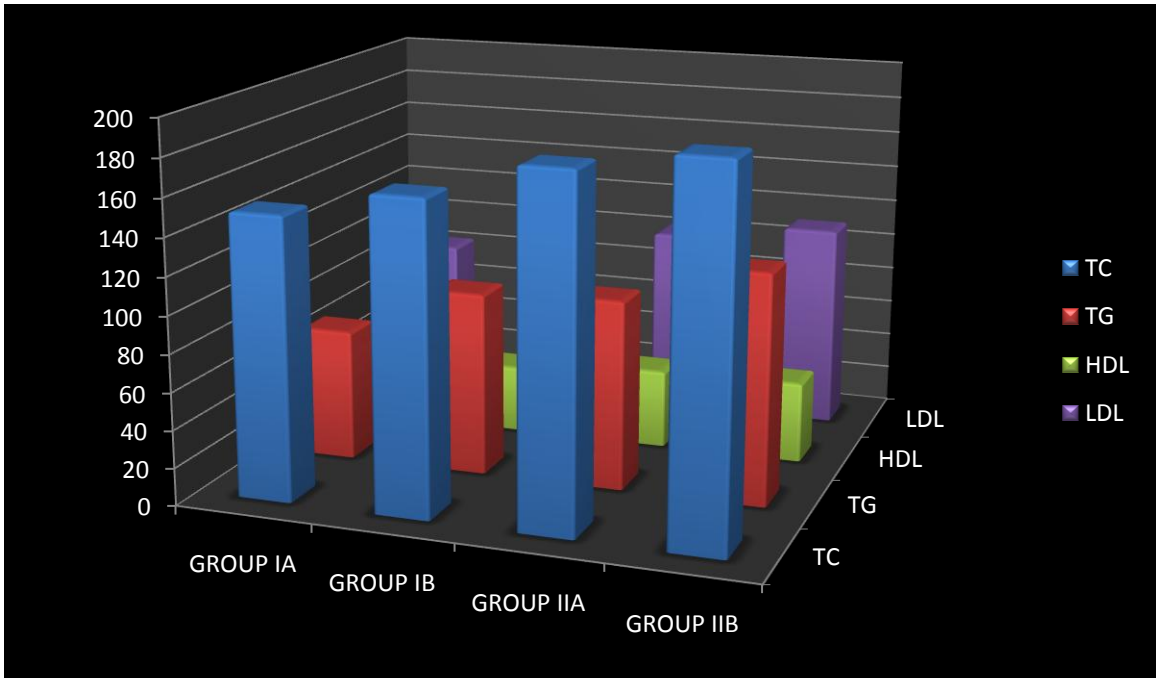


FIGURE II : COMPARISION BETWEEN HSCRP, WH RATIO, LDL/HDL BETWEEN 4 GROUPS:

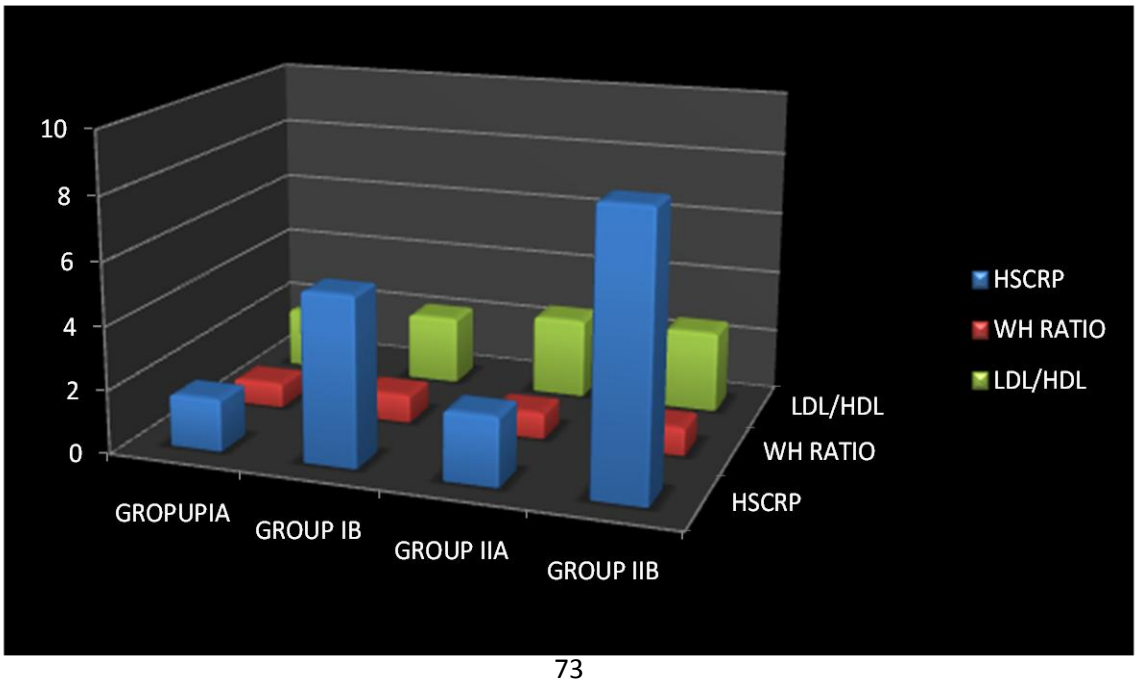


FIGURE III: PREVALENCE OF PERIODONTITIS AMONG 4 GROUPS:

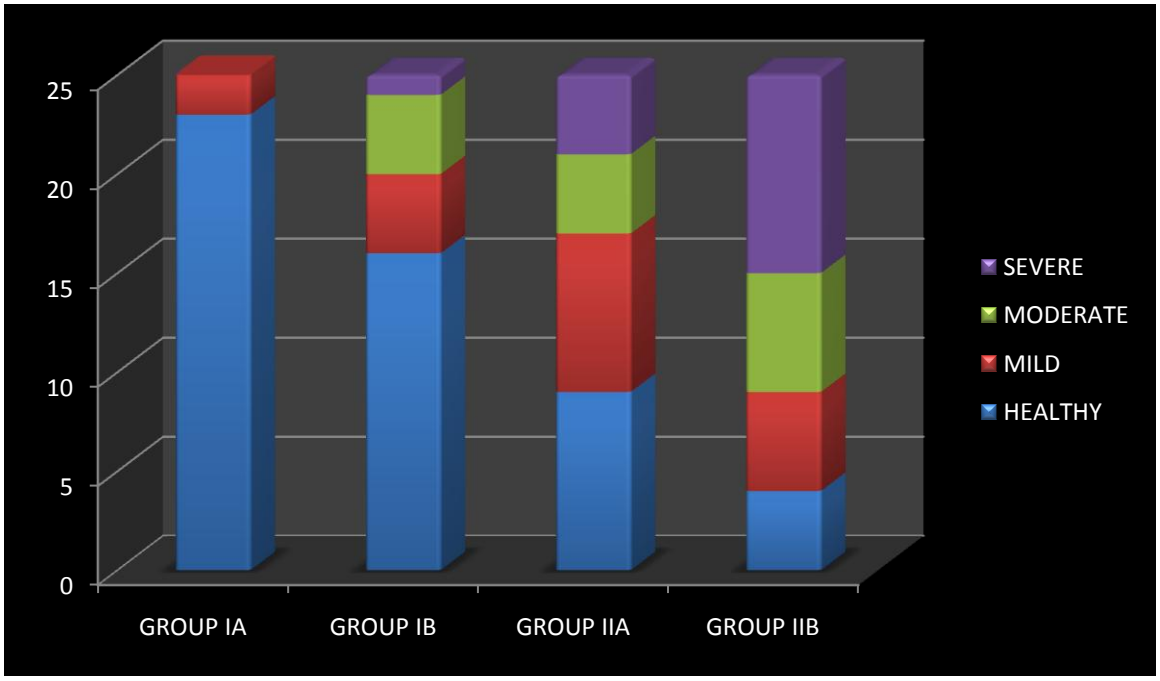


FIGURE IV: PREVALENCE OF PERIODONTITIS AMONG GENDER IN 4 GROUPS:

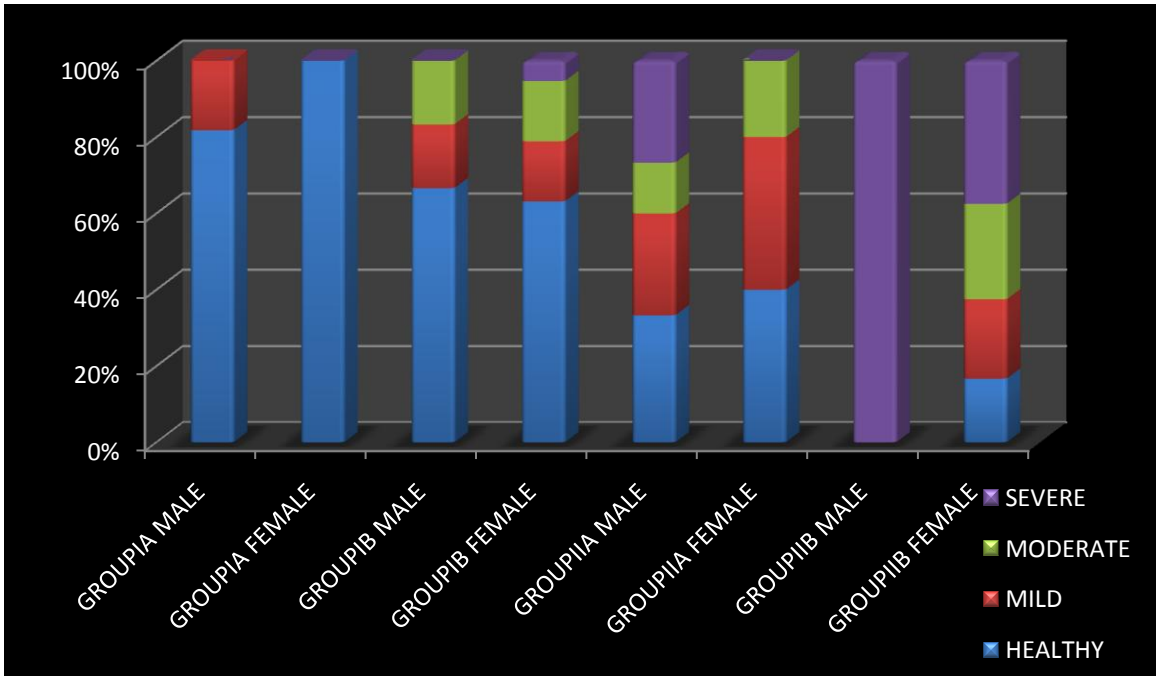


FIGURE V: PREVALENCE OF PERIODONTITIS WITH RESPECT TO TC:

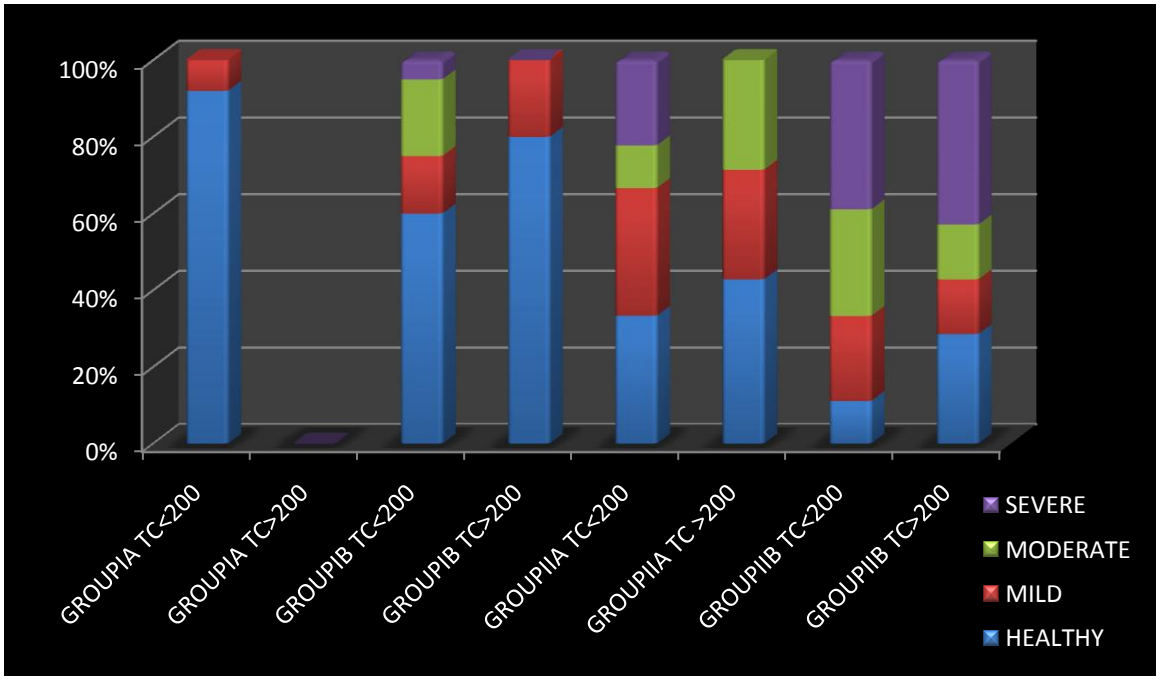


FIGURE VI: PREVALENCE OF PERIODONTITIS WITH RESPECT TO TG:

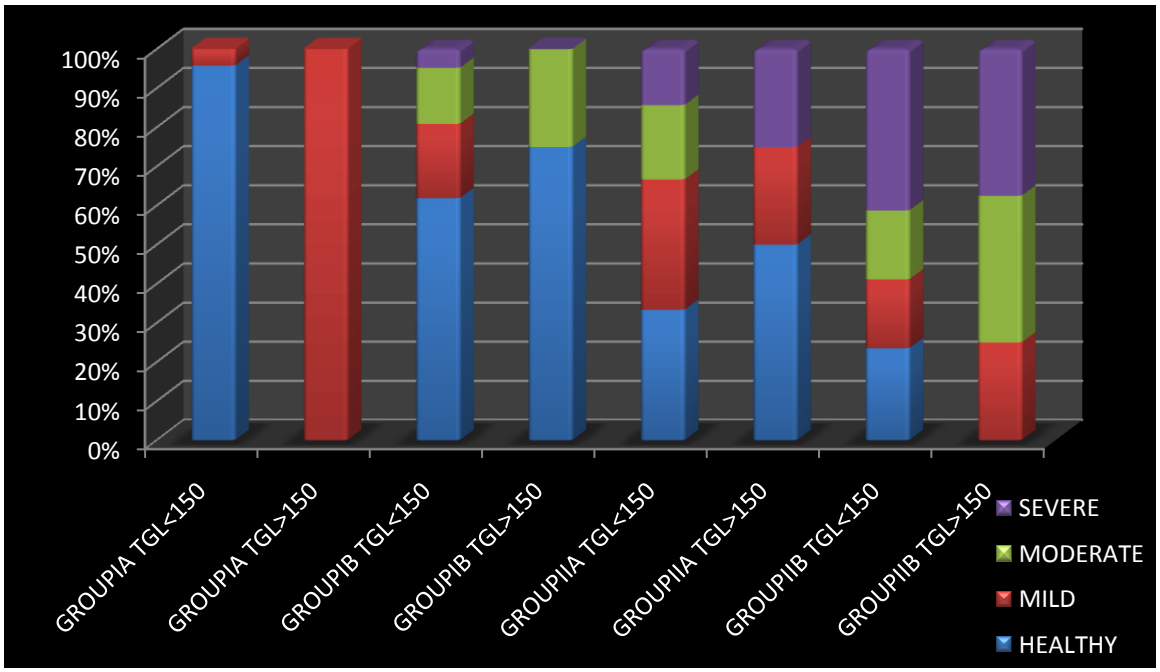


FIGURE VII: PREVALENCE OF PERIODONTITIS WITH RESPECT TO HDL:

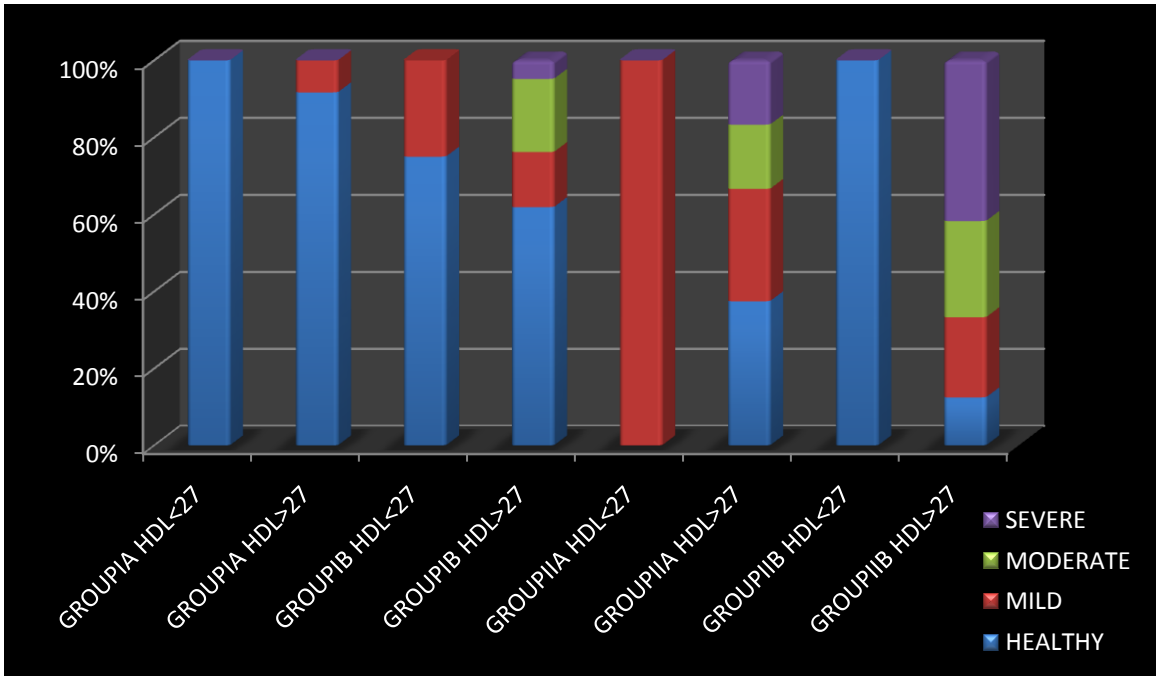


FIGURE VIII: PREVALENCE OF PERIODONTITIS WITH RESPECT TO LDL:

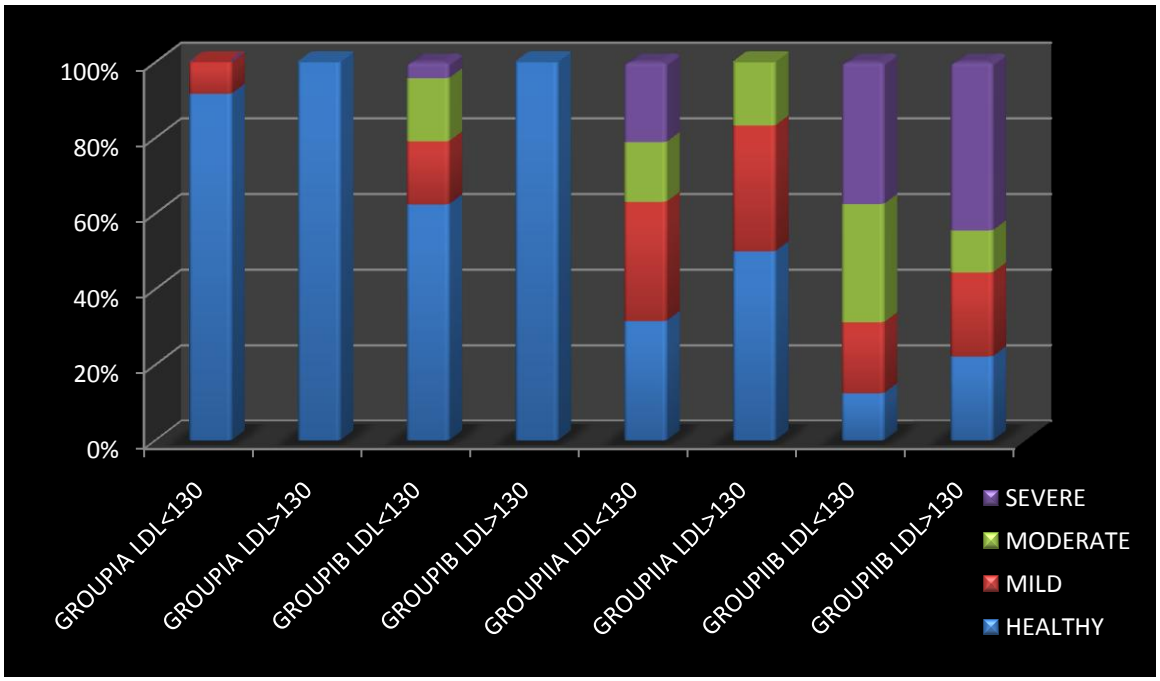


FIGURE IX: PREVALENCE OF PERIODONTITIS WITH RESPECT TO LDL/HDL RATIO:

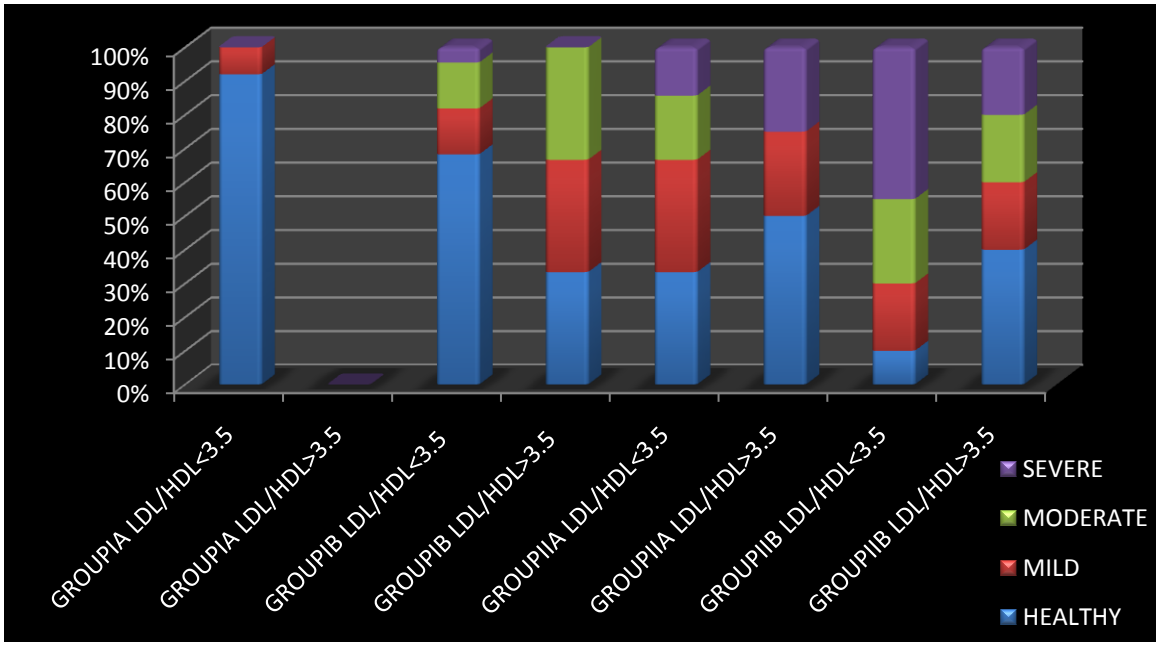


FIGURE X: PREVALENCE OF PERIODONTITIS WITH RESPECT TO HSCRP:

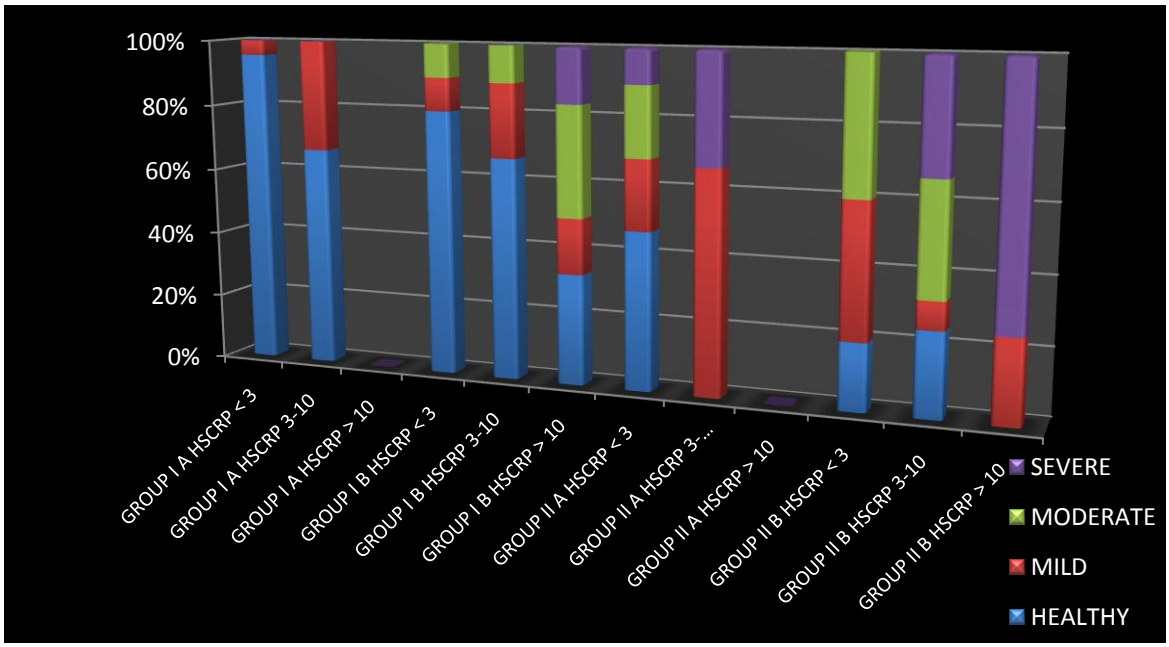
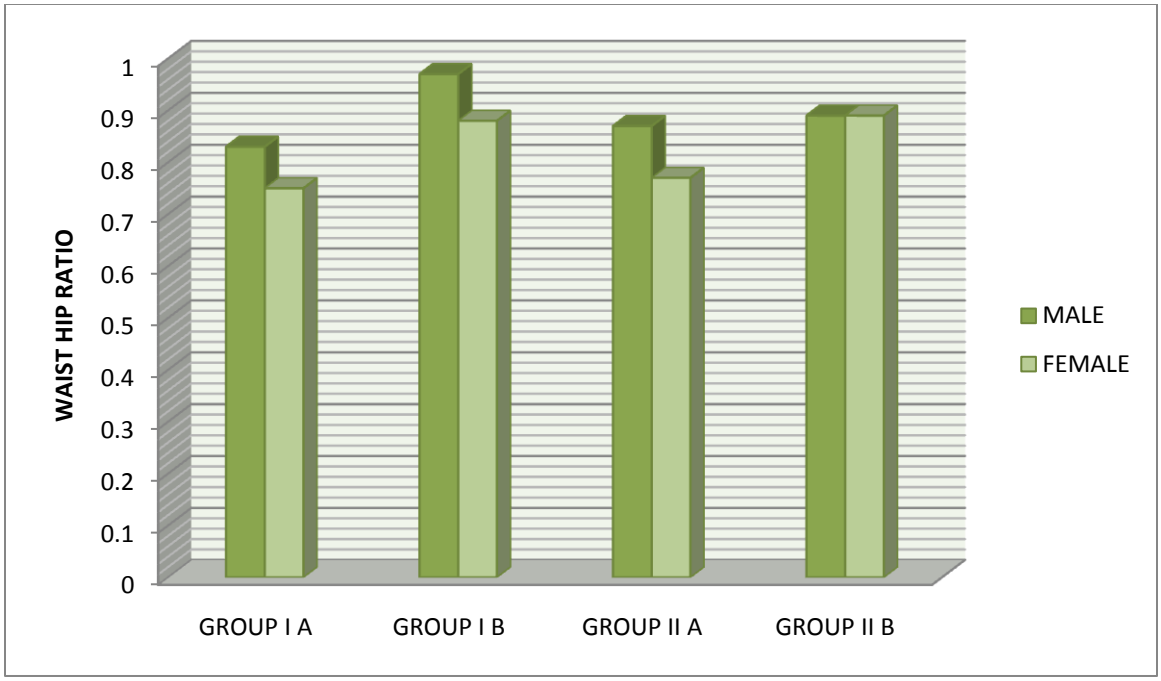


FIGURE XI : WAIST HIP RATIO



SUMMARY AND CONCLUSION

A total of 100 subjects were included in the study. The subjects were divided into 2 groups based upon their age – 18 to 34 years and 35 to 59 years and further subdivided into 2 groups based on the BMI. A complete history was taken to rule out any other systemic diseases. A thorough periodontal examination was done which included plaque index, bleeding index, probing depth and clinical attachment levels. Clinical examination included measurement of BMI and waist hip ratio as indicators of obesity.

Biochemical investigations included estimation of serum high sensitivity C reactive protein, total cholesterol, triglycerides, low density lipoprotein and high density lipoprotein.

The results showed that there was a positive correlation between body mass index and waist hip ratio with clinical attachment levels but the results obtained were not statistically significant. This could be partly explained by the relatively smaller number of sample size included in the study. Futher studies including larger sample sizes may be conducted in obese patients adjusting for age, socioeconomic status and gender to evaluate the true relationship between obesity and periodontitis.

Furthermore serum lipid profile was predominantly within the reference range in the study population. Hence it was difficult to draw conclusion between the lipidaemic status of the subjects for the severity of peridontitis.

Finally there was a significant association between serum CRP levels with clinical attachment level in younger patients whose BMI was greater than 23. This may explain the fact that periodontitis may increase systemic inflammation in conjunction with obesity.